# General Medicine & Surgery

- everythingyou need to prepareforexams
- diagrams, flowcharts, colour coding and summary boxes
- includes all core topics for medical finals kfoundationyears
- peer-reviewed with the latest guidelines

**Rebecca Richardson**
**& Ricky Ellis**

Cardiology
Endocrinology
Gastroenterology
Hepato-pancreato-biliary
Haematology
Immunology & allergy
Neurology
Renal
Respiratory
Surgical principles
The acute abdomen
GastroIntestinal surgery
The breast
Vascular surgery
Urology
Critical illness
Emergency presentations
Rheumatology
Trauma & orthopaedics

7

# General Medicine & Surgery

## medical student revision guide

> *"I like the unique style ... It is simple, engaging and easy to read, whatever your learning preferences (I particularly like the colour coding!). It also covers topics comprehensively, making it not only a great aide-memoire but also a very useful everyday book on the ward."*

From the Foreword by
Professor Farah Bhatti OBE
Consultant Cardiothoracic Surgeon

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# Foreword

A book such as this takes time, effort and dedication from all the contributors who do this alongside their already busy clinical work. It is heartening to see this commitment to supporting the education and advancement of the next generation of doctors and allied health professionals.

I like the unique style which is based on Dr Richardson's personal notes whilst a medical student. It is simple, engaging and easy to read, whatever your learning preferences (I particularly like the colour coding!) It also covers topics comprehensively, making it not only a great aide-memoire but also a very useful everyday book on the ward. The judicious use of references (e.g. NICE guidelines and BMJ Best Practice) also enables readers to look into subjects in depth if needed, without affecting the flow of the text.

Once again, I commend the authors on their remarkable efforts and wish everyone who picks up this book well in their careers.

*Professor Farah Bhatti OBE*
*Consultant Cardiothoracic Surgeon*

# Preface

**I remember it well**. Countless evenings sat at my desk, desperately trying to work out what I needed to know, and how I was going to know it. Hours spent trudging through textbooks, soaking up line after line of information, only to be left certain that after a week on my new placement, it would all be forgotten. **Medical school is tough**. The vast volume of knowledge that must be acquired and retained, to achieve the standards expected of a safe and successful doctor, is a daunting task. But as you will find at every stage in your medical career, **help is always available if you know where to look**. For you, I hope this book can be your help. I present it as your trusted companion, a loyal friend that will stand by your side through good times and bad, something to turn to when you need **guidance, reassurance, or simply just a place to start!**

There is no end of resources available for medical students, all designed to make the journey from student to doctor that little bit easier. For me, I found this wealth of information to be just as much of a curse as it was a blessing. Not only was I faced with the overwhelming task of learning it all, but I first had to find and dissect the parts that were relevant to me. **Wouldn't it be easier if everything I needed to know was in the same place?** The simple answer – yes. And that is how I started to write this book.

My first goal when creating the *Medical Student Revision Guides* was to bring together **all the key topics needed for medical school exams and life as a junior doctor** in one readily available place. I did this through summarising a variety of recognised resources, including textbooks, articles and clinical guidelines. This has been **supplemented by the expert knowledge of specialists** in each field, who have reviewed each chapter to ensure it is accurate and reflects the most up-to-date guidance.

My second goal was to do everything I could to help you remember this information. I appreciate everyone is unique in the way they learn, but with most of us relying on **vision as our dominant sense**, it seemed illogical not to utilise its power. As such, I have specifically designed this book with an **extensive use of colour, diagrams and summary tables**, to create a resource that is visually striking and a refreshing change from your current textbooks. The informal 'notes-style' layout and dedicated column that allows for your own annotations on each page, are features that I hope make the content feel **more accessible and easier to engage with**.

It is my sincere hope that you find this book useful, whether it be as your comprehensive revision resource, or a quick reminder of a condition that you come across on the ward. **I am forever grateful for any feedback** that can help me to better help you, so please do leave a review with your honest thoughts.

I wish you luck with your exams, and all the best for your future careers.

*Rebecca Richardson*

**Disclaimer**

It is important to note that this book is designed as a revision tool and aide-memoire. It is not intended to give an in-depth understanding of each condition, but rather to focus on the key points that often appear in undergraduate exams. It should not be solely relied upon in clinical situations; please always check the most current and local guidelines before implementing management or administering any treatment.

Every attempt has been made to ensure that the most up-to-date information has been included at the time of writing this book. However, due to the continuously evolving nature of the medical profession, and with variations in clinical practice between hospital Trusts, this cannot be guaranteed. It is therefore advised that you correlate these notes to other resources, and supplement them with your own clinical encounters, to ensure a complete learning experience. Readers should also ensure that they learn all elements of their own medical school curriculum, regardless of whether they are covered in this book.

# Acknowledgements

I must firstly say a huge thank you to the team at Scion Publishing for making this book a reality. A special mention to Jonathan and Clare, for your tireless work overcoming layout issues and design problems, to ensure the end product was everything I had hoped it would be.

Secondly, I would like to thank my co-author, Ricky. Your advice and guidance have been invaluable in this process. Your wealth of experience in the field of medical education has undoubtedly benefited the book and its readers, and remains an inspiration for my future work.

My thanks are extended to all those who have contributed time and expertise as a chapter reviewer. I will be eternally grateful that you could see the potential in my work in its early stages and gave up your own time to help me achieve it. The abundance of knowledge and experience you have brought is priceless and will underpin the learning of many future doctors.

Finally, to my loved ones. To Mum and Dad, for the years of selflessness and sacrifice that allowed me the opportunities to achieve all that I have today. To my brother, Chris, whose artistic talent never fails to amaze me, and has inspired the covers and diagrams of both my books. To Nana, Gran, Grandad and Margaret, for the timeless wisdom and unconditional love that only grandparents can provide. And to Martin, for your endless patience, support and encouragement in all that I do.

Thank you.

# About the authors

**Rebecca Richardson** is a junior doctor currently working in the East Midlands. After graduating from the University of Nottingham as one of the top students in her year, with first class honours, she became passionate about helping others to follow in her footsteps. Her revision notes have already helped hundreds of medical students across the UK prepare for their exams. Rebecca has continued her mission to support students and trainees through creating content for a variety of medical education platforms, as well as running regular virtual teaching sessions to help students practise for their clinical examinations.

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# General abbreviations

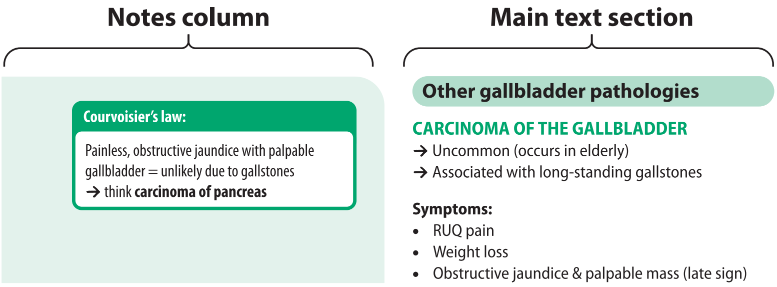
Each chapter begins with a set of abbreviations specific for that chapter.

**2ww** – 2 week wait
**a.** – Arterial
**ABCDE** – Airways, Breathing, Circulation, Disability, Exposure
**ABG** – Arterial blood gas
**ABX** – Antibiotics
**AXR** – Abdominal X-ray
**BB** – Beta-blocker
**BD** – Twice a day
**BG** – Blood glucose
**BMI** – Body mass index
**BP** – Blood pressure
**ca** – Cancer
**Ca** – Calcium
**CBT** – Cognitive behavioural therapy
**CCB** – Calcium channel blocker
**CCF** – Congestive cardiac failure
**CF** – Cystic fibrosis
**CHF** – Chronic heart failure
**CI** – Contraindication
**CK** – Creatine kinase
**CKD** – Chronic kidney disease
**CMV** – Cytomegalovirus
**CN** – Cranial nerve
**CNS** – Central nervous system
**CPAP** – Continuous positive airway pressure
**CPR** – Cardiopulmonary resuscitation
**Cr** – Creatinine
**CRP** – C-reactive protein
**CRT** – Capillary refill time
**CSF** – Cerebrospinal fluid
**CT** – Computed tomography
**CV** – Cardiovascular
**CVA** – Cerebrovascular accident
**CVD** – Cardiovascular disease
**CXR** – Chest X-ray
**D&V** – Diarrhoea and vomiting
**d** – Day
**DBP** – Diastolic blood pressure
**DDx** – Differential diagnosis
**DM** – Diabetes mellitus
**DOAC** – Direct oral anticoagulant
**DRE** – Digital rectal examination
**DVLA** – Driver and Vehicle Licensing Agency
**Dx** – Diagnosis
**ECG** – Electrocardiogram
**ECT** – Electroconvulsive therapy
**EEG** – Electroencephalogram
**eGFR** – Estimated GFR
**ENT** – Ear, nose and throat
**EPO** – Erythropoeitin
**ESR** – Erythrocyte sedimentation rate
**FBC** – Full blood count
**FHx** – Family history
**G&S** – Group & save
**GA** – General anaesthetic
**GCS** – Glasgow Coma Score/Scale
**GFR** – Glomerular filtration rate
**GI** – Gastrointestinal
**GP** – General practitioner
**h** – Hour
**Hb** – Haemoglobin
**HF** – Heart failure
**HIV** – Human immunodeficiency virus
**HR** – Heart rate
**HTN** – Hypertension
**ICP** – Intracranial pressure
**Ig** – Immunoglobulin
**IHD** – Ischaemic heart disease
**IM** – Intramuscular
**inc.** – Including
**IV** – Intravenous
**IVDU** – Intravenous drug user
**Ix** – Investigation
**JVP** – Jugular venous pressure
**LFT** – Liver function test
**LHS** – Left-hand side
**LMWH** – Low molecular weight heparin
**LOC** – Loss of consciousness
**m** – Month
**mcg** – Microgram
**MCS** – Microscopy, culture and sensitivity
**MDT** – Multidisciplinary team
**MI** – Myocardial infarction
**min** – Minute
**MND** – Motor neurone disease
**MRI** – Magnetic resonance imaging
**Mx** – Management
**NBM** – Nil by mouth
**NSAID** – Non-steroidal anti-inflammatory drug
**N&V** – Nausea & vomiting
**OCD** – Obsessive–compulsive disorder
**OD** – Once a day
**O/E** – On examination
**OT** – Occupational therapist
**PCR** – Polymerase chain reaction
**PE** – Pulmonary embolism
**PET** – Positron emission tomography
**PHx** – Past history
**PNS** – Peripheral nervous system
**PO** – (per ora) Orally
**PR** – Per rectum
**PRN** – Pro re nata (as required)
**pulm.** – Pulmonary
**QDS** – Four times a day
**RA** – Rheumatoid arthritis
**RBC** – Red blood cell
**RFT** – Renal function test
**RHS** – Right-hand side
**r/o** – Risk of
**R/V** – Review
**re** – Regarding
**RF** – Risk factor
**RR** – Respiration rate
**s** – Second
**SALT** – Speech and language therapy
**SBP** – Systolic blood pressure
**SC** – Subcutaneous
**SD** – Standard deviation
**SE** – Side-effect
**SL** – Sublingual
**SLE** – Systemic lupus erythematosus
**SOB** – Shortness of breath
**SSRI** – Selective serotonin reuptake inhibitor
**Sx** – Symptoms
**TB** – Tuberculosis
**TCA** – Tricyclic antidepressant
**TDS** – Three times a day
**TFT** – Thyroid function test
**TIA** – Transient ischaemic attack
**TNM** – Tumour, nodes, metastases
**Tx** – Treatment
**U&Es** – Urea & electrolytes
**USS** – Ultrasound scan
**UTI** – Urinary tract infection
**VBG** – Venous blood gas
**vit** – Vitamin
**VTE** – Venous thromboembolism
**W** – Week
**WBC** – White blood cell
**WCC** – White cell count
**WHO** – World Health Organization
**y** – Year

# How to use this book

The underlying principle of this book is to present information in a way that is **eye-catching, clear and easy to remember.** This page will explain some of the **key layout features** that have been used to achieve this.

**1. Notes column** – each page is divided into a main text section, and a tinted notes column. The notes column is used to house additional information, and to provide space for your own notes, should you wish to make any.



**Courvoisier's law:**

Painless, obstructive jaundice with palpable gallbladder = unlikely due to gallstones
→ think **carcinoma of pancreas**

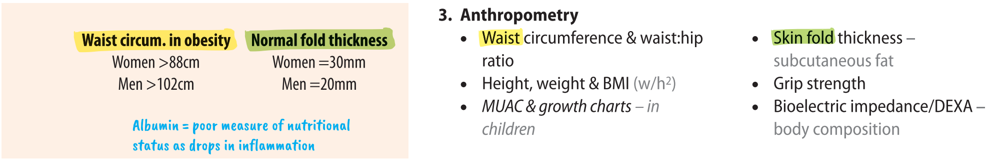
**Other gallbladder pathologies**

**CARCINOMA OF THE GALLBLADDER**
→ Uncommon (occurs in elderly)
→ Associated with long-standing gallstones

**Symptoms:**
- RUQ pain
- Weight loss
- Obstructive jaundice & palpable mass (late sign)

**2. Text colour**
- chapter coloured text – used to expand on a point / provide extra information
- grey text – used for less important information
- red text – used for red flags and emergency points
- blue text – used for extra points / annotations

**3. Highlighting** – words/phrases that have been highlighted are linked to extra information. Look for another highlight of the same colour on the page to find this information. *The below example uses green highlighting to link 'Skin fold' to the additional information 'Normal fold thickness'.*



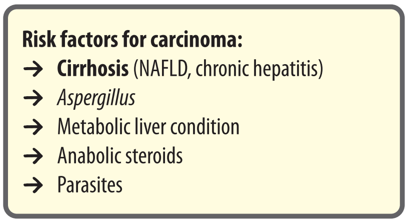
**Waist circum. in obesity**
Women >88cm
Men >102cm

**Normal fold thickness**
Women =30mm
Men =20mm

Albumin = poor measure of nutritional status as drops in inflammation

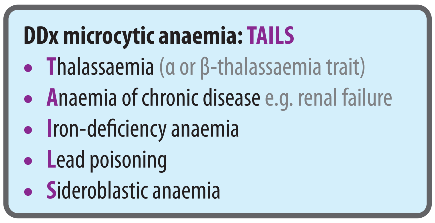
**3. Anthropometry**
- Waist circumference & waist:hip ratio
- Height, weight & BMI (w/h²)
- *MUAC & growth charts – in children*
- Skin fold thickness – subcutaneous fat
- Grip strength
- Bioelectric impedance/DEXA – body composition

**4. Box colours** – a variety of different boxes are used to display information.
- **Yellow tinted box** – for triggers / risk factors, complications & prognosis
- **Blue tinted box** – for differential diagnoses
- **Red box** – for red flags / emergency points
- **Chapter coloured box** – for all other types of information



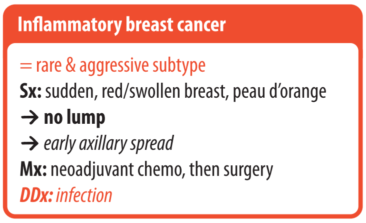
**Risk factors for carcinoma:**
→ **Cirrhosis** (NAFLD, chronic hepatitis)
→ *Aspergillus*
→ Metabolic liver condition
→ Anabolic steroids
→ Parasites

Yellow-tinted



**DDx microcytic anaemia: TAILS**
- Thalassaemia (α or β-thalassaemia trait)
- Anaemia of chronic disease e.g. renal failure
- Iron-deficiency anaemia
- Lead poisoning
- Sideroblastic anaemia

Blue-tinted



**Inflammatory breast cancer**

= rare & aggressive subtype
**Sx:** sudden, red/swollen breast, peau d'orange
→ **no lump**
→ *early axillary spread*
**Mx:** neoadjuvant chemo, then surgery
***DDx:*** *infection*

Red

# 01

# CARDIOLOGY

Acute coronary syndrome ....... 2
Chronic stable angina ....... 4
Heart valve disease ....... 5
Infective endocarditis ....... 7
Congestive cardiac failure ....... 8
Acute pulmonary oedema ....... 9
Syncope ....... 10
Atrial fibrillation ....... 11
Miscellaneous cardiac conditions ....... 12

## ABBREVIATIONS

**ACEi** – Angiotensin-converting enzyme inhibitor
**ACS** – Acute coronary syndrome
**ADH** – Anti-diuretic hormone
**AEDs** – Anti-epileptic drugs
**AF** – Atrial fibrillation
**ARB** – Angiotensin receptor blocker
**AS** – Aortic stenosis
**ASD** – Atrial septal defect
**BB** – Beta-blocker
**BNP** – B-type natriuretic peptide
**CABG** – Coronary artery bypass graft
**CAD** – Coronary artery disease
**CCB** – Calcium channel blocker
**CCF** – Congestive cardiac failure
**CO** – Cardiac output
**CPAP** – Continuous positive airway pressure
**CSA** – Chronic stable angina
**CSS** – Carotid sinus syndrome
**CTD** – Connective tissue disease
**CVD** – Cardiovascular disease
**DAPT** – Dual antiplatelet therapy
**DCCV** – Direct current cardioversion
**DOAC** – Direct oral anticoagulant
**ECHO** – Echocardiogram
**EF** – Ejection fraction
**GORD** – Gastro-oesophageal reflux disease
**GTN** – Glyceryl trinitrate
**HCM** – Hypertrophic cardiomyopathy
**HF** – Heart failure
**HFpEF** – Heart failure with preserved ejection fraction
**HFrEF** – Heart failure with reduced ejection fraction
**HR** – Heart rate
**HTN** – Hypertension
**ICD** – Implantable cardiac defibrillator
**ICH** – Intracranial haemorrhage
**IHD** – Ischaemic heart disease
**ILD** – Interstitial lung disease
**IVDU** – Intravenous drug user
**JVP** – Jugular venous pressure
**LA** – Left atrium
**LAD** – Left anterior descending artery
**LBBB** – Left bundle branch block
**LOC** – Loss of consciousness
**LSBP** – Lying and standing blood pressure
**LV** – Left ventricle
**LVEF** – Left ventricular ejection fraction
**LVF** – Left ventricular failure
**LVH** – Left ventricular hypertrophy
**MI** – Myocardial infarction
**MR** – Mitral regurgitation
**MS** – Mitral stenosis
**MSK** – Musculoskeletal
**MVP** – Mitral valve prolapse
**NSTEMI** – Non-ST-elevation myocardial infarction
**OSA** – Obstructive sleep apnoea
**PCI** – Percutaneous coronary intervention
**PE** – Pulmonary embolism
**PFO** – Patent foramen ovale
**PFTs** – Pulmonary function tests
**PMC** – Percutaneous mitral balloon commissurotomy
**PND** – Paroxysmal nocturnal dyspnoea
**PPI** – Proton pump inhibitor
**PUD** – Peptic ulcer disease
**RA** – Right atrium
**RAAS** – Renin–angiotensin–aldosterone system
**RBBB** – Right bundle branch block
**RHF** – Right heart failure
**RL CCB** – Rate-limiting calcium channel blocker
**RV** – Right ventricle
**SJW** – St John's Wort
**SNS** – Sympathetic nervous system
**SOB** – Shortness of breath
**SSRI** – Selective serotonin reuptake inhibitor
**STEMI** – ST-elevation myocardial infarction
**TAVI/TAVR** – Transcatheter aortic valve implantation / replacement
**TIA** – Transient ischaemic attack
**TOE** – Transoesophageal echo
**TPR** – Total peripheral resistance
**TTE** – Trans-thoracic echo
**UA** – Unstable angina
**URT** – Upper respiratory tract
**VF** – Ventricular fibrillation
**VSD** – Ventricular septal defect
**VT** – Ventricular tachycardia

# Acute coronary syndrome

**Key differentials:**

- Coronary spasm
- Pericarditis/ myocarditis
- Aortic dissection
- MSK (costochondritis)
- PE
- Pneumothorax
- GORD
- PUD

**Risk factors**

- Smoking
- Hypertension
- Alcohol
- Diabetes mellitus
- FHx
- Previous IHD

**BEWARE ATYPICAL PRESENTATION: 'silent MI' = ACS with no chest pain**

**Instead:** syncope / pulmonary oedema / epigastric pain → in elderly/diabetics/females

**Serial troponin:** shows continued rise at 6h, **peak at 24h**

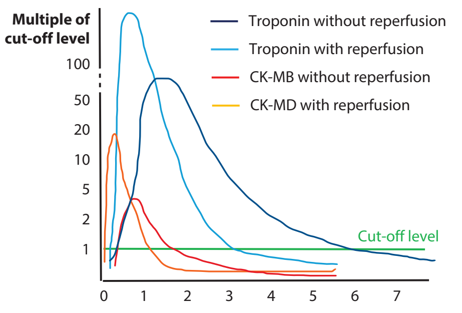


**Fig. 1.1**

**STEMI:** complete occlusion
= ST elevation **AND** ↑ troponin

**NSTEMI:** partial occlusion
= ST depression / T wave inversion **OR** normal ECG **AND** ↑ troponin

**UA:** partial occlusion
= ST depression/ T wave inversion **WITHOUT** ↑ troponin

**ST-elevation criteria:**

**>1 mm** in ≥2 contiguous* **limb leads**
OR
**>2mm** in ≥2 contiguous* **chest leads**
+ **ST depression in reciprocal leads**

**Posterior STEMI:**

**ST depression** in ≥2 chest leads **V1–4**
AND **≥0.5mm** ST elevation in any **V7–9**

*contiguous = represent same territory

→ **STEMI, NSTEMI & unstable angina** (not stable angina)

## Pathogenesis

1. **Atherosclerosis:** fatty deposition in artery wall causes plaque build-up
2. **ACS when:** Plaque rupture → Thrombosis → Vasoconstriction → Ischaemia ± distal thromboembolism

Results in a transmural or subendocardial infarct (circumferential infarct if global hypoperfusion)

## Clinical presentation

→ **Severe, *'crushing'* chest pain** (at rest)

- retrosternal, radiating to arm/neck/jaw
- **lasts >20min & NOT RELIEVED by 3× GTN sprays***

*Angina is <20min / relievable

→ **Sympathetic symptoms:** sweating, palpitations, dyspnoea, nausea

- sense of 'impending doom'

## On examination

- **Sympathetic Sx:** pallor, sweating, tachycardia
- **Myocardial impairment:** hypotension, ↑ JVP, basal creps, 3rd heart sound, pulmonary/peripheral oedema

## Investigations

1. **Bloods:** ↑ lipids, ↓ glucose, clotting
2. **Serial troponin:** on admission + 6–12h later →
3. **ECG:** within 10min of presentation
4. **Other:** CXR, ECHO, angiogram

(r/o DDx, assess prognosis & determine best Tx)

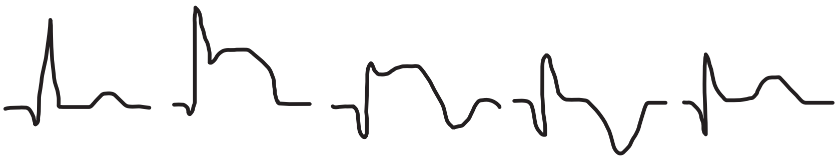
**Other causes ↑ troponin:**

- myocarditis/pericarditis
- **post-PCI**
- ventricular strain / HF
- burns/sepsis
- **PE**
- renal failure

**But these show consistently high levels**

### STEMI ECG CHANGES:

***New LBBB + chest pain = assumed STEMI***



**At onset of pain** normal sinus complex

**Within 1h** ST elevation

**Following Tx** T-wave inversion Deep Q waves

**24h later** ST normalised T-wave inversion

**Days/months** deep Q-wave (tissue death)

**Fig. 1.2**

Widespread concave ST elevation = pericarditis

### NSTEMI ECG CHANGES (IF PRESENT):

**≥0.5mm** ST depression in ≥2 contiguous* **leads**
AND **>1mm T depression** in leads with +ve QRS

**Takotsubo: 'broken heart syndrome' (stress cardiomyopathy):** appears with Sx & ECG changes of STEMI but normal angiogram

## Management[1]

| | NSTEMI/UA | STEMI |
|---|---|---|
| **Acute** | Morphine + metoclopramide (5–10mg)<br>Oxygen (if sats <90%)<br>Nitrates (GTN spray)<br>Antiplatelets (300mg chewable aspirin + loading dose ticagrelor/clopidogrel/prasugrel)<br>Anticoagulant (fondaparinux 2.5mg for 72h) | |
| | 1. **GRACE score:** assess inpatient & 6m mortality<br>2. **DAPT score:** predicts who will benefit from dual antiplatelet therapy<br>3. **Angiographic assessment**<br>4. **Revascularisation options:** PCI, CABG | First line: **primary PCI** ideally if <120min from onset (95% success)<br>**Target 150min call → balloon time**<br>Second line: **thrombolysis** (rarely used as many contraindications) |
| **Long-term** | A 1. **Lifelong Aspirin:** 75mg OD<br>A 2. **Continue 2nd Antiplatelet:** ticagrelor/clopidogrel/prasugrel (for 1y)<br>BB 3. **Lifelong Beta-Blocker:** bisoprolol (HR at 60bpm)<br>A 4. **Lifelong Atorvastatin**<br>A 5. **ACEi:** ramipril<br>6. **Treat underlying problems:** HF/HTN (BP<140/90) | |
| **Rehab** | ***Optimise physical, psychological & social functioning + stabilise/slow atherosclerosis***<br>**Patient education:** about risk factors for CVD<br>**Exercise programme:** gradual return<br>**Return to work:** usually after 2w (depends on job) | |

*Remember acute management as 'MONA(A)'*

RETURN TO DRIVING:
**Angina:** if Sx controlled
**PCI:** 1w
**CABG:** 4w
**ICD:** 6m

**GRACE score parameters to predict inpatient & 6m mortality**

1. Age
2. HR
3. SBP
4. Creatinine
5. Any CHF
6. Cardiac arrest at admission
7. ST segment changes
8. Elevated cardiac markers

Although GRACE score has good predictive value of mortality, it is often not used in practice.

Local logistics determines speed of cardiac catheterisation. STEMI have primary PCI immediately on arrival, NSTEMI and unstable angina have inpatient cardiac catheterisation as soon as feasible, prior to discharge.

**NSTEMI: dual antiplatelet therapy**
Aspirin + ticagrelor/clopidogrel/prasugrel
→ consider lower dose if high bleeding risk (HASBLED score)
→ consider stopping if going for CABG revascularisation
→ consider GI protection with PPI if >65y or Hx of GORD/PUD

## Complications of ACS

| | | |
|---|---|---|
| D | **Death** | |
| A | **Arrhythmia** | AF, VF, VT, bradyarrhythmias (VF = most common complication) |
| R | **Rupture** | Of papillary muscles / ventricular wall / septum (causes MR, VSD & LVF) |
| T | **Tamponade** | Due to ventricular wall rupture = Beck's triad |
| H | **Heart failure** | May be due to VSD = **cardiogenic shock** |
| | | |
| V | **Valve problem** | Due to papillary muscle dysfunction/rupture = **new murmur** |
| A | **Aneurysm of ventricle** | Due to weakened ventricular wall |
| D | **Dressler's syndrome** | Immune-mediated pericarditis (usually 2–3w later) |
| E | **Emboli** | From thrombus forming over damaged left ventricle wall |
| R | **Recurrence** | |

**Beck's triad:**

1. ↑JVP
2. ↓BP
3. Quiet heart sounds

**Dressler's syndrome:**

1. Fever
2. Chest pain
3. Exertional dyspnoea
4. Pericardial effusion

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[1] NICE (2020) *Acute coronary syndromes* [NG185]

# Chronic stable angina

**Risk factors**
- Hypertension
- ↑ Cholesterol
- Smoking
- FHx of premature CAD
- Diabetes mellitus

**Causes of CSA:**
- **CAD:** atheroma → thrombus
- **Valve disease***: increases workload
- **HCM****: ↓ contractility
- **Anaemia:** increased demand

*Aortic stenosis most commonly
**Hypertrophic cardiomyopathy causes LV outflow obstruction & ↑ workload

**Anatomical tests:** show degree of obstruction/plaque formation
→ *confirms Dx of CAD & gives prognostic information for consideration of revascularisation*

**Use of GTN spray:**
- sit down & 1–2 puffs
- wait 5min & if still Sx = **repeat 1–2 puffs**
- wait 5min & if still Sx = **call 999**

**Revascularisation (PCI or CABG) if significant disease:**
- Left main stem / proximal LAD disease
- 2–3 vessels disease + impaired LV function
- Limiting angina **despite optimal medical Tx**
- Large area (>10%) of myocardium at risk

**Choose PCI:**
- discrete narrowing
- <3 vessels involved
- frail patient
- comorbidities that will affect rehab
- patient choice after informed of options

**Choose CABG:**
- >3 vessels involved
- diabetic
- PHx narrowing/stents
- other cardiac pathology needing simultaneous Tx e.g. valve disease

**Episodic pain** occurring **during ↑ myocardial demand** due to ↓ perfusion

## Clinical presentation

→ **Retrosternal pain/discomfort/'tightness'**: radiating to arm/neck/jaw

1. **Occurs on exertion**
2. **Usually <10min** **can be exacerbated by cold / big meals**
3. **Relieved with rest & GTN spray** (unlike ACS)

## Classification of severity

**Canadian Cardiovascular Society (CCS) grading system**

| Grade | Description |
|---|---|
| 1 | Angina only with **strenuous exertion** e.g. rapid/prolonged activity |
| 2 | Angina with **moderate exertion** e.g. in the cold, after eating, uphill |
| 3 | Angina with **mild exertion** e.g. walking a few blocks / 1 flight of stairs |
| 4 | Angina **at rest** |

## Investigations

1. **HISTORY:** most important
2. **Examination:** find underlying causes – DM, hypercholesterolaemia, HTN, valve disease
3. **Investigation of cause:**
   → ECG – **usually normal** but may be signs of ischaemia / previous MI / arrhythmias
   → Bloods – FBC, U&Es, glucose, lipids, platelets, TFTs
   → CXR – signs of HF, r/o pulmonary disease
   → Echo – show LV function, cardiomyopathies, valve disease
4. **Further tests**
   → Exercise tolerance test – reproduce Sx and/or show ST depression & T inversion during exertion → functional test
   → CT coronary angiogram – **non-invasive** → anatomical test (to r/o CSA if pre-test probability is low)
   → Cardiac catheterisation – **invasive** → anatomical test

No need to inform DVLA if symptoms are controlled

## Management[2]

1) **Lifestyle**: diet, weight (BMI <25), exercise (30–60min per day), smoking/alcohol cessation
2) **Symptom relief:** sublingual GTN spray
3) **Anti-anginal**
   **1st line:** beta-blocker (bisoprolol/nebivolol)
   **2nd line:** CCB if BB contraindicated (amlodipine or diltiazem)
   **3rd line:** combine BB & non-rate-limiting CCB* or nitrate
   **4th line:** add nicorandil **or** ivabradine **or** ranolazine

***Rate-limiting CCB + BB = CONTRAINDICATED**

4) **Secondary prevention**
   - **Antiplatelet:** 75mg aspirin OD
   - **Statin:** atorvastatin, simvastatin, rosuvastatin
5) **Control comorbidities**
   - **HTN:** aim for BP ≤130/80
   - **Diabetes:** ACEi

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[2] NICE (2011, updated 2016) *Stable angina* [CG126]

# Heart valve disease

## Causes

| | Pathogenesis | Example |
|---|---|---|
| **Congenital** | Abnormal valve formation | Bicuspid aortic valve → aortic stenosis or regurgitation |
| **Degenerative** | Slow degeneration over time | Mitral valve prolapse |
| | Calcification (with age) | Aortic stenosis |
| | Post-MI ischaemia / papillary muscle rupture | Mitral regurgitation |
| **Infective** | Direct infection | Bacterial endocarditis |
| | Immune-mediated damage | Rheumatic fever (rare) |
| **Cardiac remodelling** | Causes dilated valve rings | Functional: mitral regurgitation<br>Pathological: connective tissue disease |

## Investigations of valve disease

1. **Hx & examination:** murmurs, signs of HF, AF
2. **ECG:** AF, ventricular hypertrophy (large R wave), atrial hypertrophy (bifid P wave)
3. **CXR:** size of heart & vessels, calcification, pulmonary oedema
4. **Echo:** dimensions of chambers, valve morphology, LV function, wall defects
5. **Doppler:** shows regurgitation (measures pressure gradient across valve)

## Medical management of valve disease[3]

1. **Slow rate of structural remodelling/deterioration:** ACEis (for mitral regurgitation)
2. **Symptom control:** diuretics (if heart failure), beta-blockers & anticoagulation (if AF/palpitations)
3. **Periodic surveillance:** if non-severe aortic stenosis (with echo)

**Tricuspid/pulmonary disease = RARE**
**Causes:** IVDU, rheumatic fever

**General principles of management:**

1. **Mild disease:** no treatment
2. **Moderate disease:** medical Tx (surgical if having concomitant cardiac surgery)
3. **Severe disease:** surgical repair/replacement (TAVI/TAVR)

## Mitral valve disease

| | Mitral stenosis | Mitral regurgitation |
|---|---|---|
| **Causes** | Rheumatic fever (rare in Western countries) | Degenerative (MVP), endocarditis, LV remodelling/dilation, Papillary dysfunction (MI) |
| **Pathogenesis** | LA can't empty = pulmonary HTN<br>→ can result in secondary RHF | LV can't fill = ↑ blood in LA & pulmonary HTN<br>Results in ↓ CO so LV works harder = LV dilation |
| **Symptoms** | **Asymptomatic if mild**<br>→ Dyspnoea & haemoptysis (pulmonary HTN)<br>→ Fatigue, weakness, peripheral oedema (RHF)<br>→ Palpitations (RA hypertrophy) | **Asymptomatic for a long time**<br>→ Dyspnoea & orthopnoea (pulmonary HTN)<br>→ Fatigue, weakness (↓ CO)<br>→ Palpitations (AF) |
| **Signs** | → Malar flush<br>→ Irregular pulse / AF<br>→ ↑ JVP<br>MID-DIASTOLIC MURMUR (loud S1 'opening SNAP') | → Irregular pulse / AF<br>→ Laterally displaced apex (LV hypertrophy)<br>PANSYSTOLIC MURMUR (soft H1) |
| **Treatment** | 1. **Percutaneous mitral balloon commissurotomy**<br>2. **Surgery:** replacement | 1. **Surgical repair or replacement**<br>(repair > replacement if favourable anatomy) |

### SURGICAL OPTIONS FOR MITRAL VALVE DISEASE[3]:

1. **Percutaneous mitral balloon commissurotomy (PMC)/valvuloplasty**
   → **1st line in mitral stenosis** if no contraindications
2. **Mitral valve repair:** repair leaflets & insert supportive ring & new chordae
   → Preferred to replacement if possible

**Contraindications for PMC:**

- Only mild MS (mitral valve area $>1.5\text{cm}^2$)
- LA thrombus
- Moderate/severe mitral regurgitation
- Severe calcification
- Severe aortic valve disease needing surgery

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[3] 2021 ESC/EACTS Guidelines for the management of valvular heart disease; *Eur. Heart J.* 43:561.

3. **Mitral valve replacement:** mechanical or biological
   - → Mechanical mitral valves at HIGHER RISK OF THROMBOSIS than mechanical aortic valves
   - → Mechanical mitral valves are potentially MORE DURABLE than bioprosthetic valves

## Aortic valve disease

| | Aortic stenosis | Aortic regurgitation |
|---|---|---|
| **Causes** | CALCIFICATION (common), congenital bicuspid valve | Endocarditis, syphilis, rheumatic fever, CTD |
| **Pathogenesis** | LV can't empty = LV hypertrophy<br>Results in LHF, angina & arrhythmia | ↑ blood in LV & ↓ CO<br>Results in LV dilation |
| **Symptoms** | → **S**yncope<br>→ **A**ngina + **A**rrhythmia<br>→ **D**yspnoea (exertional) | **Asymptomatic for a long time**<br>→ Angina<br>→ Dyspnoea |
| **Signs** | → Slow rising pulse<br>→ Narrow pulse pressure<br>*EJECTION SYSTOLIC MURMUR (radiates to neck)*<br>+ Soft S2 (AV closure)<br>+ Systolic thrill | → Collapsing pulse<br>→ Wide pulse pressure<br>→ **Quincke's sign:** nailbed pulsation<br>→ **De Musset's sign:** head bobbing<br>*EARLY DIASTOLIC MURMUR* |
| **Treatment** | **Aortic valve replacement (TAVR/TAVI)**<br>→ If symptomatic + severe stenosis on echo<br>→ If asymptomatic & severe stenosis | **Surgical valve replacement**<br>→ If significantly enlarged aorta<br>→ If severe regurgitation + LVEF <55% |

### SURGICAL OPTIONS FOR AORTIC VALVE DISEASE[3]:

1. **Surgical aortic valve replacement:** via sternotomy (usual option for **severe** aortic stenosis/regurgitation)

→ if younger, fit, healthy patient **or** need cardiac surgery for concurrent coronary artery disease

2. **Transcatheter aortic valve replacement/implantation (TAVR/TAVI):**

→ catheter through femoral artery (transfemoral) or apex of heart (transapical)

→ balloon dilation to deploy valve

→ if older/frailer (contraindications for open heart surgery)

*Those with heart valve disease often have concurrent IHD which should be treated AT SAME TIME AS VALVE OPERATION*

## Mechanical vs. biological valves

→ **Mechanical require lifelong anticoagulation:** WARFARIN (INR 2.5–3.5)

→ **Biological:** increasing durability to 10–15y & only need anticoagulation for 3m (unless needed for comorbidity like AF)

**DOACs contraindicated in mechanical heart valves**

### Cardiac catheterisation/angiography

**Procedure:**

1. **For imaging of coronary arteries:**
   Catheter through right radial/femoral artery → contrast medium injected → X-ray series taken
2. **For assessment of right heart & pulmonary artery pressures:**
   Catheter through femoral vein

**Risks:** bleeding, bruising, allergy to contrast medium, pericardial effusion/tamponade, death (very rare)

**Indications:**

1. **Diagnosis:** CAD, valve disease, cardiomyopathy, congenital heart disease, pulmonary HTN
2. **Treating conditions with a transcatheter approach:** PCI*, TAVI, closure of PFO/ASD, some cases of mitral regurgitation (using Mitraclip)

*Percutaneous coronary intervention (PCI) = using catheter + balloon & stents to open a blocked/narrow vessel

# Infective endocarditis

Infection of endocardial heart surface (especially heart valves)

## Pathophysiology

→ **Damaged endocardium** allows **bacterial adhesion**
→ Become encased in fibrin mesh → protected while multiply
→ **Form vegetations** that damage valves & chordae

**Causative organisms:** (rarely fungi)

- *Strep. viridans* (in dental/URT disease)
- *Staph. aureus* (IVDU)
- *Strep. epidermis* (in prosthetic valves)
- Enterococci
- *Strep. bovis* (in bowel pathology)
- Candida (immunosuppressed)

## Risk factors

1. **Abnormal structure + commensal organisms**
   - prosthetic valve, valve disease, congenital defect
   - commensals (skin/GI tract) enter circulation to reach valves (e.g. following dental treatment)
   - more insidious onset
2. **Normal structure + pathogenic organisms**
   - IVDU, open heart surgery, septicaemia
   - pathogenic organisms can infect normal valves
   - acute, fulminating onset

**Common valves affected**
→ usually LHS (mitral > aortic)
→ tricuspid valve in IVDU

## Clinical presentation

| | |
|---|---|
| **M** | **M**urmur & **M**icroscopic haematuria |
| **A** | **A**naemia |
| **J** | **J**aneway lesions (spots on palms) |
| **O** | **O**sler's nodes (nodules on fingers) |
| **R** | **R**oth spots (eyes) |
| **P** | **P**yrexia |
| **E** | **E**mboli |
| **N** | **N**ail splinter haemorrhages |

→ *febrile illness + new murmur*

**ANTIBIOTIC PROPHYLAXIS NO LONGER ROUTINELY GIVEN when undergoing invasive procedures or dental work.** Check with cardiologist for individual patients at high risk.

**Complications:**

1. **Systemic emboli:** brain/kidneys
2. **Valvular incompetence & CCF**
3. **Glomerulonephritis:** immune complexes deposit in kidneys

## Investigations

1. **Hx & examination:** for risk factors, fever, new murmur
2. **Bedside tests:** urine dip & ECG (haematuria, MI due to emboli)
3. **Routine bloods:** FBC, CRP/ESR, U&Es (infection, anaemia)
4. **Triple blood cultures:** from 3 different sites → **before ABX**
5. **Transthoracic (TTE) & transoesophageal echo (TOE) → diagnostic**
6. **Other imaging:** CXR & CT (signs of HF/emboli)

**TTE first, then TOE if:**

1. High clinical suspicion
2. Positive findings on TTE
3. Poor image quality
4. Suspected aortic root abscess

## Diagnosis

*Duke's criteria*

**Major criteria**

- +ve blood culture of **typical organism** at **2 separate sites**
- Echo shows endocardial involvement

**Minor criteria**

- Predisposing risk factors (prosthetic valve, IVDU)
- Fever >38°C
- Vascular phenomena (Janeway lesions/emboli)
- Immunological phenomena (glomerulonephritis/Osler's)
- +ve blood culture not meeting major criteria

**Diagnosis made if:**
→ 2 major **OR**
→ 1 major + 3 minor **OR**
→ 5 minor

## Management[4]

**MANAGEMENT: microbiology advice**

1. **IV ABX:** 6 weeks
   → Should respond in 48h (↓ fever & CRP/ESR)
   - usually penicillin-based (± gentamicin)
   - teicoplanin, vancomycin if **prosthetic valves**
2. **Surgery if:**
   - persistent bacteraemia after 72h ABX
   - heart failure results
   - valvular obstruction / unstable valve
   - conduction disturbance
   - repeated emboli

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[4] 2015 ESC Guidelines for the management of infective endocarditis; *Eur. Heart J.* 36:3075

# Congestive cardiac failure

Abnormality of structure or function causing inadequate pumping

## Pathophysiology

**Compensatory changes** occur to maintain CO & BP → leads to **hypertrophy & dilation → cardiac failure**

1. **Activated SNS:** ↑TPR & ↑HR (vasoconstriction increases afterload)
2. **Activated RAAS:** ↑TPR & fluid retention (increases preload & afterload)
3. **ADH secretion:** fluid retention (increases preload)

## Causes of heart failure

| | **Systolic HF**<br>insufficient pumping function | **Diastolic HF**<br>poor filling due to ↓compliance / impaired relaxation |
|---|---|---|
| **Ejection fraction** | **EF <40%** | **EF >50%**<br>(HF with preserved EF → HFpEF) |
| **Causes** | 1. **IHD** (CAD → MI → LV dysfunction)<br>2. Myocarditis → dilated cardiomyopathy<br>3. Cor pulmonale / COPD = RH failure | 1. Chronic hypertension<br>2. Restrictive/hypertrophic cardiomyopathy<br>3. Fibrosis/amyloidosis/sarcoidosis<br>4. Constrictive pericarditis<br>5. Haemochromatosis<br>6. Aging |

## Symptoms

| | **Right-sided failure**<br>= systemic congestion | **Left-sided failure**<br>= pulmonary congestion |
|---|---|---|
| **Symptoms** | • Dyspnoea & fatigue<br>• Peripheral oedema & ascites<br>• Hepatomegaly | • Fatigue & reduced exercise tolerance<br>• Dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea<br>• Nocturnal cough = pink, frothy sputum |
| **Signs** | • ↑JVP, hepatomegaly, ascites<br>• Pitting oedema & face engorged<br>• Pleural effusions | • **Cardiomegaly:** displaced apex<br>• **Tachycardia:** 3rd heart sound, gallop rhythm<br>• **Pulmonary oedema:** bibasal crackles |
| **Common signs** | Cyanosis, decreased BP, narrow pulse pressure, displaced apex, RV heave<br>**Cardiac cachexia:** ↑metabolic demands + ↓appetite | |

*Usually LHS & RHS HF occur simultaneously so there is overlap of symptoms*

**New York Heart Association classification of HF**

**Class I:** no limitation of activity (no dyspnoea/fatigue)
**Class II:** slight limitation (dyspnoea/fatigue on moderate activity)
**Class III:** marked limitation (only comfortable at rest)
**Class IV:** symptoms at rest

## Investigations

*falsely low BNP if on ACEi, BB, diuretic*

1. **Careful Hx & examination**
2. **Bloods:** FBC, U&Es, LFT, TFT
3. **BNP:** strong –ve predictive value (if normal = unlikely HF)
4. **ECG:** may show cause e.g. ischaemia, arrhythmia, HTN
5. **CXR:** cardiomegaly, pulmonary oedema
6. **Echo:** show structure abnormalities & measure EF → only if ECG/BNP is abnormal

→ ***Possible further tests:*** *cardiac MRI, cardiac catheterisation, stress echo*

## Management[5]

**ACUTE HEART FAILURE = emergency (due to pulmonary oedema)**

- **ABCDE:** sit patient upright, high flow $O_2$, IV diuretics (furosemide)
- Consider IV nitrates if no improvement (caution if SBP <100 / aortic stenosis)
- Consider CPAP & inotropes if needed

**CHRONIC HEART FAILURE**

1. **Lifestyle:** smoking cessation, alcohol & salt reduction, exercise, BMI optimisation
2. **Symptom relief:** diuretics (furosemide)* & med R/V (NSAIDs)
3. **Counteract maladaptation:** medications **(renal monitoring)**
4. **Consider IHD risk:** statin + aspirin 75mg if high *do not prescribe RL CCB in HF*

****daily weights if inpatient***

**Goals of treatment:**

1. Treat any underlying cause e.g. IHD
2. ↓Cardiac workload (afterload)
3. ↑Cardiac output (stroke volume)
4. Counteract maladaptation
5. **Symptom relief**

*Ramipril* *Bisoprolol*

**1st line:** ACEi + BB (ARB if ACEi not tolerated)
↓
**2nd line:** spironolactone + nitrate
↓
**3rd line:** digoxin (resynchronisation if wide QRS)

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[5] NICE (2018) *Chronic heart failure in adults* [NG106]

# Acute pulmonary oedema

Increased fluid in pulmonary interstitium

## Causes

- **LEFT VENTRICULAR FAILURE:** post-MI or IHD (most common cause)
- **Other cardiac causes:** valve disease, arrhythmias, hypertension
- **Acute respiratory distress:** trauma, drugs, malaria
- **Fluid overload**
- **Neurogenic:** head injury

## Clinical presentation

- **Dyspnoea, orthopnoea, PND** (sat up, leaning forward)
- Productive cough with pink, frothy sputum
- Distressed, pale, sweaty

## Clinical signs

- ↑ HR, ↑ RR, ↑ JVP
- Gallop rhythm (3rd heart sound)
- Fine crackles ± wheeze

## Investigations

1. **Careful Hx & examination**
2. **Bedside:** ECG (tachycardia, LVH, arrhythmias)
3. **Bloods:** FBC, U&Es, CRP, glucose, troponin, **ABG***
4. **Imaging:** CXR ± echo (clarify cause e.g. valve disease / MI)

## Management[6]

***EMERGENCY → begin treatment before investigations***

**As for acute heart failure (see above)**

- **ABCDE:** sit patient upright, high flow $O_2$ (if hypoxia)
- **IV diuretics** (furosemide / loop diuretic = 1st line)
- GTN spray x2 puffs (caution if SBP <90)
- Consider IV nitrates if no improvement (caution if SBP <100 / aortic stenosis)
- Consider CPAP & inotropes if needed

## Features on CXR

- Batwing oedema
- Kerley B & septal lines = thickened fissures
- Upper vessel engorgement (cephalisation)
- Cardiomegaly
- Fluid in costophrenic angles (small pulmonary effusions)

**Differentials:**
- Asthma/COPD
- Pneumonia
- Pneumothorax
- Interstitial lung disease

*Initially T1RF (hyperventilation) then T2RF (impaired gas exchange)

**Monitor:**
- BP, pulse
- $O_2$ sats, ABG, RR
- JVP
- Urine output
- ECG

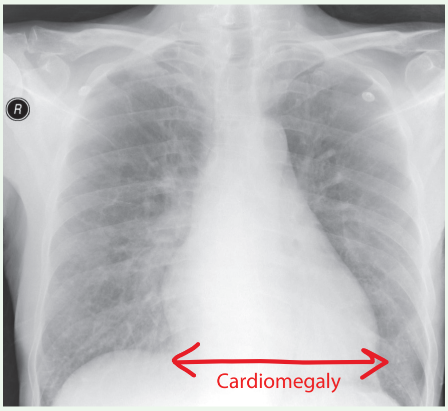

**Fig. 1.3** Pulmonary oedema CXR.

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[6] BMJ Best Practice (2021, updated 2022) *Acute heart failure*

# Syncope

**RED FLAGS in red suggest possible life-threatening cause**

Sudden, transient loss of consciousness with spontaneous & rapid resolution

**PATHOPHYSIOLOGY:** transient reduction in cerebral blood flow

### CARDIAC

- **Elderly:** sinoatrial disease, AV nodal disease / heart block
- **Young:** long QT syndrome, Brugada syndrome
- **Structural heart disease** e.g. HCM causing ventricular arrhythmias

**Symptoms:** occurs on exertion, palpitations, SOB, chest pain

### NEURALLY-MEDIATED

- **Vasovagal**
- **Situational** (cough, micturition, defecation)
- **Carotid sinus syndrome** (CSS)

**Symptoms:** light-headedness, nausea, pallor, sweating, visual disturbance

***Provoked by:*** *hot environment, pain, emotion, prolonged standing, pressure on carotid baroreceptors (CSS)*

### ORTHOSTATIC

- **Autonomic failure**
- **Volume depletion**

**Symptoms:** occurs on standing, dizziness, weakness, tachycardia, preceding melaena/haematemesis

***Causative drugs:*** *antihypertensives, diuretics, vasodilators*

**Postural drop on LSBP:**
SBP drop of ≥20
or DBP drop ≥10

**Red flag differentials:**

- Myocardial ischaemia
- Arrhythmias
- Aortic dissection
- Occult haemorrhage
- Severe hypoglycaemia
- Massive PE
- Subarachnoid haemorrhage

## Investigations[7]

- Thorough history – preceding symptoms, DHx, PMHx, FHX + RED FLAGS
- Physical examination – including lying & standing blood pressure (LSBP), HR, RR
- 12-lead ECG – check for arrhythmias / heart block / ischaemia
- Bloods – Hb (anaemia), U&Es, **troponin, glucose, D-dimer**
- CT head – if any concern of head injury / stroke / TIA
- Ambulatory ECG recording (7d)

## Management

### CARDIAC SYNCOPE

- ILR (implantable loop recorder) – placed substernal subcutaneous, under local anaesthetic
- ICD / pacemaker implant
- Anti-arrhythmic therapy (VT ablation in selected patients)

### VASOVAGAL SYNCOPE[8]

1. **Patient education:** avoid triggers (prolonged standing, warm environments, etc.)
2. **Physical counter-pressure manoeuvres:** muscle tensing / leg crossing / squatting
3. **Volume expansion:** increase salt & fluid intake (beware may be contraindicated in some patients)
4. **Medication:** fludrocortisone, midodrine → only occasionally used in the elderly

---

[7] BMJ Best Practice (2021) *Assessment of syncope*
[8] BMJ Best Practice (2021) *Neurally mediated reflex syncope*

# Atrial fibrillation

Rapid, irregular heart rhythm due to uncoordinated contraction of the atria

1. **Paroxysmal:** recurrent, sudden, self-limiting episodes of palpitations → sometimes Tx with **'pill in pocket'** e.g. flecainide
2. **Persistent:** AF >7 days → needs **beta-blocker** to rate control & **cardioversion** to 'reset' rhythm
3. **Permanent:** long-term AF → **RATE CONTROL** vs. rhythm control

## Causes

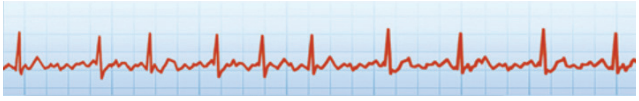
- **Cardiac:** HTN, heart failure, IHD, valve disease
- **Respiratory:** PE, chest infection, lung cancer
- **Systemic:** sepsis, thyrotoxicosis, ↑alcohol/caffeine, ↓electrolytes

**Causes:** Mrs **SMITH** has AF
**S**epsis
**M**itral valve stenosis
**I**HD
**T**hyrotoxicosis
**H**ypertension

## Investigations*

1. **Systems examination:**
    - Irregularly irregular pulse
    - Signs of underlying disease, e.g. murmur/HF
2. **12-lead ECG:** if normal do 24h ambulatory ECG

**Fig. 1.4**



1. Absent P waves 2. Irregular R-R interval 3. Rapid rate

3. **Bloods:**
    - FBC, TFTs, U&Es, LFTs, glucose
    - Alcohol levels (if suspicious)
4. **CXR/ECHO:** investigate cause

*Consider app on mobile phone, to enable rhythm recording by patient during symptoms

## Management[9]

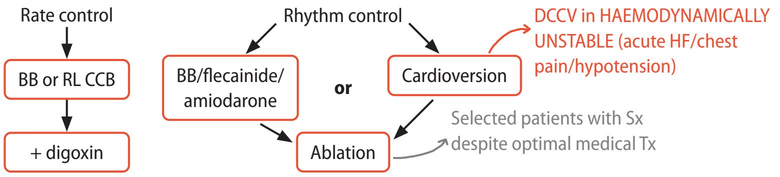
DO NOT COMBINE BB + RL CCB

### A) RATE & RHYTHM CONTROL

* NOT digoxin as monotherapy unless immobile

1. Rate control: target HR **60–80** → beta-blocker (**or** RL CCB if BB contraindicated) (often combined with digoxin*)
2. Rhythm control: if **new onset** <48h, **reversible cause, HF** → flecainide/amiodarone **or** DCCV (direct current cardioversion)

Long-term side-effects: hepatotoxic, phototoxic, thyrotoxic, lung fibrosis


Rate control
BB or RL CCB
+ digoxin
Rhythm control
BB/flecainide/ amiodarone
or
Cardioversion
DCCV in HAEMODYNAMICALLY UNSTABLE (acute HF/chest pain/hypotension)
Ablation
Selected patients with Sx despite optimal medical Tx

### B) STROKE PREVENTION (ANTICOAGULATION)

1. Assess risk with $CHA_2DS_2$-VASc score: anticoagulate females if score ≥2 or men if ≥1

→ **DOAC** (rivaroxaban, apixaban, edoxaban, dabigatran) = more predictable & ↓ICH risk

→ **Warfarin** (if mechanical valve/mitral stenosis or low eGFR) = **needs close monitoring**

2. Assess bleeding risk with ORBIT* score: ≥3 needs closer anticoag. monitoring

### C) LIFESTYLE

- Weight loss, diet, exercise
- ↓alcohol, caffeine, smoking
- Optimise RFs: BP, renal function, etc.

---

[9] NICE (2021) *Atrial fibrillation* [NG196]

**Complications**
- **STROKE**
- ↓LV function
- Peripheral embolism
- Vascular dementia
- ↓quality of life

**Symptoms**
- SOB
- Syncope
- Palpitations
- Chest pain

**Warfarin:** vit K antagonist (reversible)
↑INR: NSAIDs, cranberry juice, SSRIs, ABX
↓INR: AEDs, rifampicin, barbiturates, SJW, smoking
*NB: warfarin = teratogenic* **INR aim: 2–3**

| **CHADSVaSc score** | | |
|---|---|---|
| **C** | Congestive HF | 1 |
| **H** | HTN | 1 |
| **$A_2$** | Age >74y | 2 |
| | Age 65–74y | 1 |
| **D** | Diabetes | 1 |
| **$S_2$** | Previous stroke/TIA | 2 |
| **Va** | Vascular disease | 1 |
| **Sc** | Sex category = female | 1 |

| **5 Predictors of ORBIT score** | |
|---|---|
| Age ≥75y | 1 |
| eGFR <60 | 1 |
| Treatment with antiplatelet | 1 |
| Bleeding history | 2 |
| Hb <130 (females) <140 (males), ↓haematocrit or Hx of anaemia | 2 |

***ORBIT score** now recommended by NICE over HASBLED score to calculate bleeding risk for those with AF

**Follow-up**
- In 1y to check for Sx of AF
- Annual R/V of stroke & bleed risk

**Safety-net**
- Signs of MI/stroke (LOC, severe chest pain/SOB, dizzy)
- If HR >150 or SBP <90

Advise may need to inform DVLA (if symptomatic)

For **elective electrical cardioversion** (not emergency): ANTICOAGULATE before with LMWH or 3 weeks other agent

# Miscellaneous cardiac conditions

## Coronary artery bypass graft → most common cardiac surgery

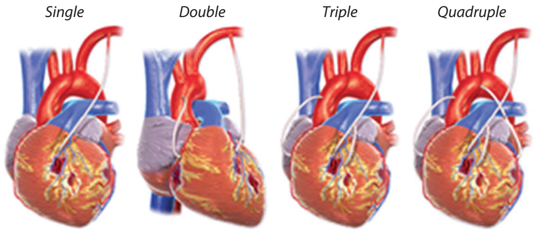


**Fig. 1.5** Types of CABG.

### INDICATIONS:

**Bypass narrowings in coronary arteries if:**

- Left main stem stenosis
- Diabetes with multivessel disease
- 2–3 vessel disease & impaired left ventricle

### TECHNIQUES:

1. **On pump:** arrest heart & use cardiopulmonary bypass machine
2. **Off pump:** performed on beating heart → careful positioning & stabilisers

**Gold standard conduit:**

Left internal mammary artery (LIMA) attached to left anterior descending (LAD)

**Can also use other arteries/veins:**

- great saphenous vein
- radial artery
- right internal mammary artery

## Cardiac trauma

### TYPES:

1. **Blunt trauma:** deceleration injuries (e.g. road traffic accident)
2. **Penetrating trauma:** e.g. anterior stab wound (usually right ventricle damaged)

**What to do in a cardiac trauma**

→ **Ix:** echo (for haemopericardium), ECG ± troponin

→ **Tx:** **emergency clamshell thoracotomy** if witnessed cardiac arrest

**Complications of cardiac trauma:**

- Silent arrhythmias
- Cardiac contusion
- Cardiac wall rupture

## Atrial myxoma → most common primary tumour of heart

### SYMPTOMS: secondary to mass

→ Intermittent syncope

→ SOB

→ Fever, malaise, cachexia

### MANAGEMENT: surgical resection

→ Minimally invasive **right thoracotomy**

affects women aged 30–40y

**LOCATION:** **80% in left atrium** → may hear **'succession splash'** as 'plops' in & out of ventricle

## Constrictive pericarditis → chronic pericarditis causes thick & fibrous pericardium & constriction

### SYMPTOMS:

→ Progressive dyspnoea

→ Fatigue/weakness

→ Peripheral oedema

→ Pleural effusions

### MANAGEMENT: pericardiectomy

→ Surgical resection of pericardium between the 2 phrenic nerves

→ Usually via **sternotomy incision**

**Causes of constrictive pericarditis:**

- TB
- Cardiac surgery
- Post-MI (rare)

## Acute pericarditis → acute inflammation of the pericardium

### SYMPTOMS:

→ Pleuritic chest pain

→ Pericardial rub + tachypnoea + tachycardia

± non-productive cough, SOB, flu Sx

### INVESTIGATIONS:

1. ECG = **saddle ST elevation globally**
2. Bloods
3. Transthoracic echo to r/o myocarditis

### MANAGEMENT: NSAID + colchicine

**Causes of acute pericarditis:**

- Post-MI (Dressler's)
- Viral infection / TB
- Hypothyroidism
- CTD
- Trauma

Pain on inspiration & relief sitting forward

## Myocarditis → group of disorders causing non-ischaemic inflammation of the myocardium

### SYMPTOMS: commonly preceded by viral illness

- Fatigue
- Dyspnoea
- Chest pain (in 35%)
- Palpitations/arrhythmia
- Congestive heart failure:
  - orthopnoea, ↑ JVP, ↑ HR

### INVESTIGATIONS:

- 12-lead ECG – non-specific ST/T wave abnormalities
- CXR – may show pulmonary oedema if CHF
- CK-MB, troponin – elevated
- B-type natriuretic peptide – elevated
- Echo – global motion abnormalities
- Endomyocardial biopsy – inflammatory infiltrate

### COMPLICATIONS:

- Arrhythmias
- Dilated cardiomyopathy
- Fulminant heart failure
- Cardiogenic shock / sudden cardiac death

### MANAGEMENT[10]:

**Haemodynamically stable:**

- supportive care + treat underlying cause

**Haemodynamically stable + evidence of LHF:**

- ACEi + beta-blocker ± diuretic ± nitrate + treat underlying cause

**Haemodynamically unstable:**

- IV arterial vasodilator + invasive haemodynamic monitoring ± inotrope

+ Anticoagulation if arrhythmia or evidence of LV thrombus

**Differentials:**

- ACS
- Pericarditis

**Risk factors:**

- HIV
- autoimmune disease
- drugs (anti-epileptics, penicillins, digoxin, lithium, heavy metals)
- smallpox vaccine
- preceding viral illness
- peripartum/postnatal

*Other causes of dilated cardiomyopathy: Idiopathic, genetic, drugs, stress (takotsubo), post-viral*

**Specific Tx of underlying causes:**

**Autoimmune/hypersensitivity:** steroids

**Giant cell myocarditis:** steroids + other immunosuppressant

*Avoid NSAIDs & alcohol if history of myocarditis*

## Pulmonary hypertension → proliferation and remodelling of the small pulmonary arteries causing increased pulmonary vascular resistance (PVR)

### SYMPTOMS:

- Fatigue
- Dyspnoea
- Peripheral oedema

### SIGNS:

- Loud 2nd heart sound
- Tricuspid regurgitation murmur
- Cyanosis

### INVESTIGATIONS:

- CXR – enlarged main pulmonary artery & hilar vessels
- ECG – RV hypertrophy (tall R wave, small S wave in V1)
- Transthoracic Doppler echo – estimates pulmonary artery pressure
- Right heart catheterisation – measures mean pulmonary arterial pressure
- Nocturnal oximetry – >70% patients desaturate during sleep
- 6-minute walk test – estimate of disease severity

**Complications of pulmonary hypertension:**

- RV failure
- Tachyarrhythmias

**Diagnosis if mean pulmonary arterial pressure >25mmHg at rest**

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[10] BMJ Best Practice (2021) *Myocarditis*

## CAUSES:

| Cause of pulmonary hypertension | Specific investigations |
|---|---|
| **Idiopathic** | Diagnosis of exclusion |
| **Lung disease** e.g. COPD, ILD, OSA | ABG, PFTs, high resolution CT |
| **Chronic embolic/thrombotic disease** | V/Q scan, CTPA |
| **Connective tissue disease** e.g. scleroderma, SLE, RA | ANA, anti-centromere antibodies, rheumatoid factor |
| **Cardiac shunts** e.g. atrial/ventricular septal defect | Transoesophageal Doppler, echo |
| **Portal hypertension** in liver disease | LFTs, abdo USS |

→ Refer to pulmonary hypertension centre for consideration of secondary pulmonary vasodilator therapies

## MANAGEMENT[11]: management of underlying cause if present

1. **General supportive therapy:** supervised exercise training, reduced sodium intake **plus** oral anticoagulants (warfarin)
   ± diuretics if fluid overload
   ± oxygen if hypoxic
   ± digoxin if supraventricular arrhythmias
2. **Calcium channel blockers:** in idiopathic pulmonary hypertension

→ *2nd line options: prostanoids, endothelin receptor antagonists (bosentan), phosphodiesterase inhibitors (sildenafil)*

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[11] BMJ Best Practice (2021) *Idiopathic arterial hypertension*

# ENDOCRINOLOGY

# 02

Diabetes mellitus....16
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## ABBREVIATIONS

**Abs** – Antibodies
**ACEi** – Angiotensin-converting enzyme inhibitor
**ACTH** – Adrenocorticotrophic hormone
**ADH** – Antidiuretic hormone
**AKI** – Acute kidney injury
**Ald** – Aldosterone
**ALP** – Alkaline phosphatase
**ARB** – Angiotensin receptor blocker
**ARDS** – Acute respiratory distress syndrome
**BG** – Blood glucose
**BNP** – B-type natriuretic peptide
**Ca** – Calcium
**CAH** – Congenital adrenal hyperplasia
**Carb** – Carbohydrate
**CBG** – Capillary blood glucose
**CF** – Cystic fibrosis
**CKD** – Chronic kidney disease
**CRH** – Corticotrophin-releasing hormone
**CV** – Cardiovascular
**CVD** – Cardiovascular disease
**DA** – Dopamine
**DHEA(-S)** – Dehydroepiandrosterone (sulfate)
**DKA** – Diabetic ketoacidosis
**DM** – Diabetes mellitus
**DPP4** – Dipeptidyl-peptidase 4
**FNA** – Fine needle aspiration
**FSH** – Follicle-stimulating hormone
**GC** – Glucocorticoid
**GH** – Growth hormone
**GLP1** – Glucagon-like peptide 1
**HHS** – Hyperglycaemic hyperosmolar state
**HPA axis** – Hypothalamic–pituitary–adrenal axis
**HPT** – Hyperparathyroidism
**HRT** – Hormone replacement therapy
**ICP** – Intracranial pressure
**IGF** – Insulin-like growth factor
**IHD** – Ischaemic heart disease
**KB** – Ketone bodies
**LA** – Long-acting
**LH** – Luteinising hormone
**MC** – Mineralocorticoid
**NA** – Noradrenaline
**Na** – Sodium
**OGTT** – Oral glucose tolerance test
**OSA** – Obstructive sleep apnoea
**Osm** – Osmolality
**PCOS** – Polycystic ovarian syndrome
**$PO_4$** – Phosphate
**PSA** – Prostate-specific antigen
**PTH** – Parathyroid hormone
**RAI** – Radioactive iodine
**SA** – Short-acting
**SGLT2** – Sodium-glucose transport protein 2
**SLE** – Systemic lupus erythematosus
**SNS** – Sympathetic nervous system
**SSRI** – Selective serotonin reuptake inhibitor
**T1DM** – Type 1 diabetes mellitus
**T2DM** – Type 2 diabetes mellitus
**TPO(Ab)** – Thyroid peroxidase (antibodies)
**TRAb** – Thyroid receptor antibodies
**TSH** – Thyroid-stimulating hormone
**vit D** – Vitamin D

# Diabetes mellitus

→ 4.7 million people (1 in 15) in UK with DM
→ INCREASING PREVALENCE

| | Type 1 (8%) | Type 2 (90%) |
|---|---|---|
| **Pathogenesis** | Autoimmune beta cell destruction<br>= **absolute insulin deficiency** | Gradual beta cell decline due to prolonged hyperglycaemia<br>= **relative insulin deficiency + insulin resistance** |
| **Age of onset** | Peak **12y** (can be any age) | Peak in **70s** (getting younger) |
| **Associations** | • Autoimmune condition (islet Abs)<br>• FHx (50% twin concordance) | • Obesity, sedentary, age<br>• FHx (90% twin concordance) |
| **Symptoms** | Rapid onset: **often present with DKA**<br>• polydipsia, polyuria<br>• weight loss / thin<br>• fatigue, infections | Gradual onset: **often no Sx / present with complications**<br>• polyuria, polydipsia<br>• may lose weight, but **overweight** to start with<br>• fatigue, infections |
| **Treatment** | Lifelong insulin | Lifestyle, oral medication, insulin, bariatric surgery |

## Other types of diabetes mellitus (2%)

1. **Genetic** e.g. MODY (mature onset diabetes of young) – presents like T2DM but in <25y → monogenic inheritance
2. **Gestational DM** – usually temporary
3. **Secondary DM**
   - Pancreatic failure: pancreatitis, CF, cancer
   - Endocrine: Cushing's, acromegaly
   - Drugs: corticosteroids, beta-blockers, thyroid hormone

## Diagnosis

→ Symptoms + 1 positive lab test **OR**
→ Asymptomatic + 2 positive lab tests

*If HbA1c 42–48 suggests pre-diabetes*
*→ do fasting glucose test*

**OGTT = 75g glucose & wait 2h*

| | **Normal** | **Impaired** | **Diabetes** |
|---|---|---|---|
| **Fasting** mmol/L | <6.1 | 6.1–6.9 | **≥7.0** |
| **OGTT*** mmol/L | <7.8 | 7.8–11.0 | **≥11.1** |
| **HbA1c** mmol/mol | <42 | 42–47.9 | **≥48** |

**DO NOT USE HbA1c for Dx if:**

- Child / suspect T1DM
- Symptoms <2m
- Acute illness, anaemia, CKD
- Pregnancy
- Diabetes-inducing medications

*↳ If diagnosed, screen for target organ damage: fundoscopy, urine dip & BP, U&Es, PNS exam*

**Evaluating glucose control:**

- Finger prick = capillary blood glucose
- HbA1C = reflects 3–4m glucose control – check every 6m
- Continuous monitoring e.g. freestyle LIBRE / Dexcom

**HbA1c targets:** individualised
T1DM: **<48**
T2DM (lifestyle ± single agent): **<48**
T2DM (gliclazide or 1 agent or insulin): **<53**

## Acute complications of DM

**DKA (T1DM):** insufficient insulin causes compensatory metabolic changes to ↑ glucose (fatty acids oxidised to KBs)

**Symptoms:** N&V, abdo pain, leg cramps, hyperventilation (Kussmaul), confusion, drowsiness, collapse
→ *causes of death = cerebral oedema, hypokalaemia, ARDS, AKI, cardiac arrhythmia*

**Management**[1]**:** see *Chapter 16: Critical illness*

1. **IV fluids** 0.9% saline IV (1L in 1h)
2. **High dose, fixed rate insulin** 0.1 units/kg/h (continue until cap. ketones <0.6)
3. **Potassium replacement:** early (from 2nd bag fluids)
4. **Glucose replacement:** 10% dextrose when glucose <14mmol/L
5. **Careful monitoring (may need HDU):** glucose, ketones, VBG, U&Es, neuro status

**Diagnosis of DKA:**

1. Blood glucose **>11mmol/L** or known DM
2. Capillary ketones **>3mmol/L** or urinary +++
3. Venous pH **<7.35** or $HCO_3^-$ <15mmol/L

***= metabolic acidosis with ↑ anion gap***

**Triggers of DKA:**

- new diagnosis / non-compliance
- concurrent illness / **SURGERY**
- binge drinking / starvation

**NB: continue patient's normal long-acting SC insulin while on IV insulin**

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[1] Diabetes UK (2021) *Management of diabetic ketoacidosis in adults*

**HHS (T2DM):** severe hyperglycaemia causes hyperosmolar state WITHOUT SIGNIFICANT KETOSIS → leads to SEVERE DEHYDRATION

**Symptoms:** onset over 3–14d
- Osmotic diuresis / polydipsia + signs of dehydration
- Nausea & vomiting
- Neuro signs (confusion, seizures, aphasia, hemianopia)
- ↓ Consciousness with ↑ osmolality

**Complications:** seizures, cerebral oedema, MI/CVA, DVT/PE, foot complications

**Management**[2]**:** see *Chapter 16: Critical illness*
1. **IV fluids** 0.9% saline IV (over 24h to rehydrate)
2. **Fixed rate insulin***: 0.05 units/kg/h = HALF DOSE DKA
3. **$K^+$ replacement:** if $K^+$ drops <5.5mmol/L
4. **Prophylactic anticoagulation:** LMWH for full duration of admission

*only once glucose falling by <5mmol/L/h

Metabolic acidosis may be present if dehydration leads to **AKI**

**Diagnosis of HHS:**
1. Blood glucose **>30mmol/L**
2. Capillary ketones **<3mmol/L**
3. Venous pH **>7.3** or $HCO_3^-$ >15mmol/L
4. Osmolality **>320 mosmol/kg**

**Triggers of HHS:**
- poor control / ↑ glucose intake
- illness / infection / MI
- drugs (thiazides, steroids, BBs)

**HYPOGLYCAEMIA:** blood glucose <4mmol/L in diabetics (otherwise <3.5mmol/L with symptoms)

**Symptoms:**
- **Autonomic:** sweating, tremor, palpitations
- **Neurogenic:** confusion, aggression, anxiety, drowsiness, seizures

**Management:**
- **If conscious:** 15–20g fast-acting carb → repeat if CBG <4 in 10min (up to 3x) → long-acting carb once glucose >4
- **If unconscious:** 1mg IM glucagon **or** 100ml 20% glucose IV → long-acting carb once glucose >4

IV glucose is better as can be repeated & effective in malnourished / liver disease / alcoholic binge

**Once hypo is treated:** identify cause/RFs & address these to avoid future episodes

**Triggers of hypoglycaemia in DM:**
- SE of sulphonylureas & insulin therapy
- ↓ food, ↑ exercise, ↑ insulin
- alcohol

## Chronic complications of DM

### MICROVASCULAR

- **Nephropathy:** ↑ ×5 risk dialysis or transplant
  - BP & glycaemic control = KEY
  - **Start ACEi/ARB or SGLT2 if microalbuminaemia → target BP <130/80**

Avoid SGLT2 in T1DM as can cause ketoacidosis

- **Retinopathy/maculopathy:**
  - **Retinopathy:** no retinopathy (R0) → background (R1) → pre-proliferative (R2) → proliferative (R3)
  - **Maculopathy:** no maculopathy (M0) maculopathy (M1)
- **Neuropathy:** risk of neuropathic ulcers & Charcot joint
  - **Peripheral:** symmetrical glove & stocking distribution → amitriptyline/duloxetine/gabapentin/pregabalin if painful
  - **Autonomic:** postural hypotension, tachycardia, ↓ or ↑ sweating, gastroparesis, bladder dysfunction, erectile dysfunction
  - **Diabetic amyotrophy:** progressive, painful muscle wasting (quadriceps & shoulders)

### MACROVASCULAR

- **CVD / stroke / vascular dementia:** ↑ ×2 risk stroke
- **IHD/HTN:** ↑ ×3–4 risk MI
- **Peripheral vascular disease:** ↑ ×20 amputation (gangrene)

**If not nephropathy, BP targets are the same as normal:**
→ <140/90 CBPM or <135/85 HBPM

**RFs for chronic complications of DM**
- **long DM duration**
- **poor control**
- smoking
- HTN
- pregnancy

**Annual diabetic review:**

→ **Glycaemic control:** med R/V & compliance
→ **Retinopathy screening**
→ **Nephropathy screen:** BP, urinalysis, U&Es, Alb:Cr
→ **Foot checks & care:** early referral if needed
→ **Assess CVD risk (QRisk):** statin/antiplatelet if needed
→ **Education/advice:** smoking, contraception/ pregnancy

Alb:Cr = albumin–creatinine ratio (needs to be early morning sample)

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[2] Diabetes UK (2012) *Management of the hyperosmolar hyperglycaemic state (HHS) in adults with diabetes*

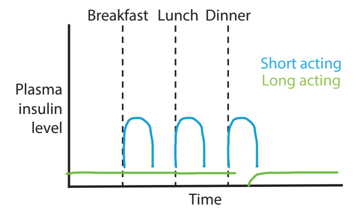


**Fig. 2.1** Action of SA & LA insulin.

### CAUTION PRESCRIBING INSULIN

Ensure you have:
- the correct type (many similar names)
- the correct dose (always write UNITS in full)
- the correct times

### Disadvantages of insulin:

- weight gain
- hypos
- regular monitoring
- injections

**Mx complications/RFs:**
1. BP = <140/90 (if nephropathy <130/80)
2. Statin if QRISK >10% or presence of microvascular disease
3. ACEi if microalbuminuria

**Sick day rules:**
1. Continue normal insulin / oral hypoglycaemic
2. Check BG 4 hourly / more often
3. Encourage fluids (sugary drinks if unable to eat)

## Management of T1DM: INSULIN

**SA** = short acting **LA** = long acting

1. **Basal-bolus:** 1 or 2 LA injections **PLUS** SA with each meal
   - Closer to natural insulin secretion pattern
   - More flexible with food/exercise → CARB COUNTING
2. **Biphasic / twice daily:** 2 doses of mixed insulin (with breakfast & dinner)
3. **Insulin pump:** via cannula → basal rate set + bolus dose with meals
   - Avoids multiple injections each day

### TYPES OF INSULIN:

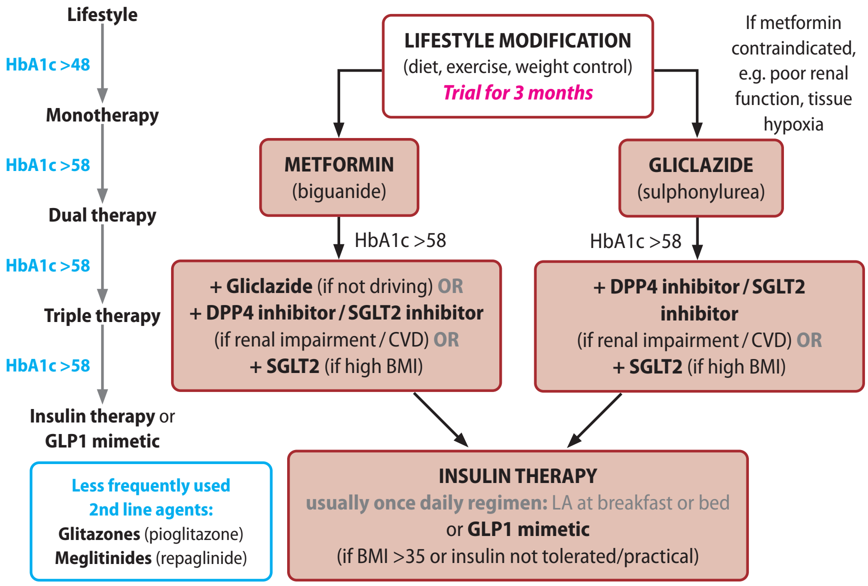
| Type | Peak | Example |
|---|---|---|
| **Rapid acting** | 1–2h | NovoRapid, Humalog |
| **Short acting** | 2–4h | ActRapid, Humulin S |
| **Intermediate acting** | 5–8h | Insulatard, Humulin I |
| **Long acting** | Relatively peakless | Lantus / Levemir / Tresiba |

## Management of T2DM[3]

**LIFESTYLE:** aim for HbA1c <48 (6.5%)
1. **Diet:** low glycaemic index carbs, ↓ fat, ↑ fibre
2. **Weight loss:** calorie restriction AND exercise (20–30min/d)
3. **Reduce smoking/alcohol**

**Education to enable self-management is key:**
1. Dietitian access
2. Websites / support groups
3. Importance of compliance / risk of complications

### PHARMACOLOGICAL



**LIFESTYLE MODIFICATION** (diet, exercise, weight control) *Trial for 3 months*

If metformin contraindicated, e.g. poor renal function, tissue hypoxia

**METFORMIN** (biguanide)

**GLICLAZIDE** (sulphonylurea)

HbA1c >58

**+ Gliclazide** (if not driving) OR **+ DPP4 inhibitor / SGLT2 inhibitor** (if renal impairment / CVD) OR **+ SGLT2** (if high BMI)

HbA1c >58

**+ DPP4 inhibitor / SGLT2 inhibitor** (if renal impairment / CVD) OR **+ SGLT2** (if high BMI)

**INSULIN THERAPY** **usually once daily regimen:** LA at breakfast or bed or **GLP1 mimetic** (if BMI >35 or insulin not tolerated/practical)

**Less frequently used 2nd line agents:** **Glitazones** (pioglitazone) **Meglitinides** (repaglinide)

**Fig. 2.2**

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[3] NICE (2015, updated 2022) *Type 2 diabetes in adults* [NG28]

## Medication side-effects

| Hypoglycaemic | Advantages | Disadvantages | |
|---|---|---|---|
| **Metformin** (**biguanides**) | ↓ CV risk<br>no weight gain | GI upset (bloating/diarrhoea)<br>not if eGFR <35 | **Stop metformin if**<br>eGFR <30 or Cr >150 (as risk lactic acidosis) |
| **Gliclazide (sulphonylurea)** | rapid effect<br>cheap | hypos<br>**weight gain** | |
| **Glitazones** | low risk of hypos<br>improve lipid profile & ↓ CVD risk | **weight gain**<br>bone fractures<br>↑ risk fluid retention | **Glitazones contraindicated in:**<br>CCF, elderly, bladder cancer |
| **SGLT2 inhibitors** | **weight loss**<br>reduce SBP & ↓ CVD risk<br>good in renal disease | UTIs / candidiasis<br>euglycaemic ketoacidosis | |
| **Sitagliptin (DDP4 inhibitors)** | low risk of hypos | nausea & acute pancreatitis | |
| **GLP1 mimetics** | **weight loss**<br>↓ CV risk if CVD | nausea & acute pancreatitis | |

# Pituitary disorders

**Mass effects of pituitary adenoma:**

- Bitemporal hemianopia = from pressure on **optic chiasm**
- Ocular palsies = from pressure on **cavernous sinus** (CN 3,4,6)
- Headache from ↑ ICP
- Altered appetite/thirst/sleep = from pressure on **hypothalamus**
- **Hypopituitarism** from destruction of functional tissue
- **Hyperprolactinaemia** from stalk effect

**'The stalk effect'**

→ **Dopamine** released from hypothalamus reaches pituitary gland **via pituitary stalk**
→ Normally dopamine **INHIBITS** prolactin
→ **Damaged/compressed stalk** = ↓ DA = ↓ inhibition = **HYPERPROLACTINAEMIA**

→ *Secretory prolactinoma **plus** compression effects = MASSIVE PROLACTIN ↑ (>10,000)*

**Hypopituitarism:**

- Sx = vague & non-specific
- General Mx = HORMONE REPLACEMENT
- Ix & Tx of specific cause

**DDx hypopituitarism:**

| Pituitary | Hypothalamic |
|---|---|
| • **Non-functioning adenoma**<br>• Metastatic tumour<br>• Surgical resection of gland<br>• Apoplexy (vascular insult) | • Craniopharyngioma<br>• Infarction<br>• Infection<br>• Sarcoid |

The pituitary gland sits below the hypothalamus & optic chiasm

## Pituitary adenoma: BENIGN

1. **Microadenoma** <1cm
2. **Macroadenoma** >1cm = mass effects
   - **Functioning:** secrete hormones (70–80%)
   - **Non-functioning:** not secretory (20–30%)

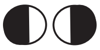

**Fig. 2.3** Visual fields in bitemporal hemianopia.

**(GH-RH)** Growth hormone-releasing hormone
**(TRH)** Thyrotropin-releasing hormone
**(CRH)** Corticotropin-releasing hormone
**(GnRH)** Gonadotropin-releasing hormone
**(SRIF)** Somatotropin release inhibiting factor / somatostatin inhibitor
**(DA)** Dopamine

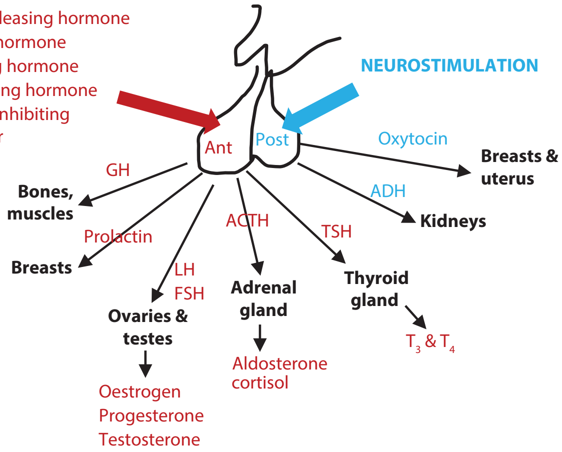

**Fig. 2.4** Functions of the pituitary gland.

## Hypopituitarism

Typical order of hormone loss ↓

| Hormone lacking | Clinical effects | Management |
|---|---|---|
| **GH** | **Children:** short stature<br>**Adults:** no Sx / abnormal body composition / fatigue | GH analogue |
| **LH/FSH** (↓ sex hormones) | **Children:** delayed puberty<br>**Adults:** no Sx / hypogonadism / amenorrhoea | Testosterone (males) or HRT (females) |
| **TSH** (↓ $T_3$/$T_4$) | Hypothyroidism (cold, weight loss, tired) | Thyroxine |
| **ACTH** (↓ cortisol) | Lethargy, weight loss, ↓ glucose, ↓ Na | Hydrocortisone/prednisolone |
| **PROLACTIN** | Failed lactation | – |
| **ADH** | Diabetes insipidus (polyuria/polydipsia) | Desmopressin |

### INVESTIGATIONS:

1. **History & examination:** including visual fields & cranial nerves
2. **Pituitary hormones:**
   - All except prolactin: LOW/normal
   - PROLACTIN
     → 500–1000: (micro-prolactinoma) **or** (non-functioning tumour compressing stalk) **or** (other cause of ↑ prolactin*) →
       - Renal impairment
       - Antipsychotics
       - Pregnancy/OCP

     → >1000: (micro-prolactinoma **AND** stalk compression) **or** (macroprolactinoma)
3. **Effector gland hormones:** LOW ($T_3$/$T_4$, FSH*, IGF-1, 9am cortisol)

*FSH should be high if post-menopausal as ↓ oestradiol (no more follicles)

4. **Stimulation tests:** LOW / no response
   - **For ACTH deficiency:** insulin stress test (IST)
   - **For acromegaly:** OGTT → GH suppression
   - **For non-pituitary cause of adrenal disease:** Synacthen test
5. **Imaging:** CT/MRI – determine pathology

**When given insulin, blood glucose drops → hypoglycaemia triggers ACTH release** – no ↑ ACTH suggests deficiency

**When given glucose in OGTT, GH should be suppressed**
No suppression suggests acromegaly (check IGF-1 levels)

## Secretory/functional adenomas

→ *can produce >1 hormone*

| Type of adenoma | Hormonal effect | Symptoms | Treatment |
|---|---|---|---|
| **Prolactinoma** 35%<br>(F:M = 5:1) | HYPERPROLACTINAEMIA<br>↑ basal prolactin | • Galactorrhoea (+ female amenorrhoea)<br>• ↓ Fertility, ↓ libido, hypogonadism<br>• **Complications:** osteoporosis | DA agonist*<br>• carbergoline<br>• bromocriptine<br>*SE: pulmonary fibrosis (need CXR) |
| **GH-secreting** 20% | ACROMEGALY/GIGANTISM<br>↑ IGF-1 & glucose | • ↑ **Size** hands/feet slow onset<br>• Coarse facies, wide-spaced teeth<br>• Vision loss<br>• Tired, **headache**, sweaty, ↓ libido<br>• **Complications:** DM, HTN, OSA | 1. Surgery: transsphenoidal<br>2. Radiotherapy<br>3. Medical<br>• DA agonist<br>• somatostatin analogue |
| **ACTH-secreting** 10% | CUSHING DISEASE<br>↑ cortisol | • ↑ Weight, DM, ↓ libido<br>• Hair, striae, hump | |
| **TSH-secreting** rare | HYPERTHYROIDISM<br>↑ $T_{3/4}$ | • ↓ Weight, fatigue<br>• Hot, sweaty, palpitations | |

*Raised TSH & T4 usually indicates poor compliance with LT4 treatment*

LH-/FSH-secreting adenoma = VERY RARE

# Adrenal disease

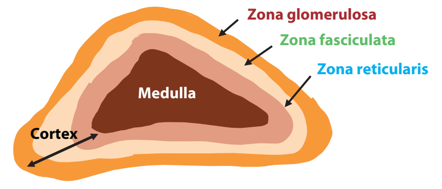

**Fig. 2.5** Adrenal gland structure.

**→ Adrenal cortex produces steroid hormones**

| Zones of the adrenal gland | | |
|---|---|---|
| Zona Glomerulosa | **Mineralocorticoids**<br>**(aldosterone)** | **$Na^+$ & $K^+$ balance**<br>= BP regulation |
| Zona Fasciculata | **Glucocorticoids**<br>**(cortisol)** | **Response to stress**<br>= glucose metabolism, inflammation |
| Zona Reticularis | **Androgens**<br>**(DHEA, DHEA-S)** | **Stimulate masculinisation** |
| Medulla | Adrenaline<br>Noradrenaline | **Response to stress**<br>= stimulate SNS |

## Addison's disease

*→ 1° adrenal insufficiency due to autoimmune destruction of cortex*

**↓ ALL STEROID HORMONES**
–ve feedback = **↑ ACTH & renin** (& CRH)

*NB 2° adrenal insufficiency if withdraw long-term steroids because long-term ACTH suppression*
*→ ONLY LOW CORTISOL (aldosterone = normal)*

### CAUSES OF GLAND DESTRUCTION:

→ **Autoimmune**
→ TB
→ Surgery
→ Severe sepsis
→ Adrenal mets

### CLINICAL FEATURES:

| **Symptoms** = non-specific | **Signs** |
|---|---|
| • Weight loss<br>• Malaise/myalgia/weakness<br>• Fatigue/depression<br>• Abdo pain / N&V<br>• Syncope | • Pigmentation (↑ ACTH = ↑ melatonin)<br>• Postural hypotension (↓ aldosterone)<br>• Dehydration (↑ urea & Cr) (↓ aldosterone)<br>• **↑ $K^+$ & ↓ $Na^+$** (↓ aldosterone)<br>• **↓ Glucose** (↓ cortisol) |

**Addisonian crisis:**

- Hypotension & tachycardia
- Severe weakness
- Abdo pain, N&V
- **Hypoglycaemia**, confusion

*→ Illness/injury can be a trigger*

**Sick day rules:** double dose hydrocortisone/ prednisolone when unwell

### INVESTIGATIONS:

1. **U&Es:** ↑ $K^+$ & ↓ $Na^+$, ↑ urea & creatinine
2. **Serum ACTH & cortisol:** ↑ ACTH, ↓ cortisol, ↓ renin
3. **SHORT SYNACTHEN TEST:** 250mcg Synacthen given
   - measure serum cortisol at 0 & 30min
   - positive test = **cortisol doesn't ↑ over** 420nmol/L
4. **Adrenal autoantibodies**
5. **Adrenal imaging:** USS/CT

### MANAGEMENT[4]:

1. **ACUTE CRISIS**
   → 100mg IV hydrocortisone + 1L 0.9% IV saline
   → ± dextrose if low glucose

2. **LONG-TERM:** lifelong hormone replacement
   → **Glucocorticoid (GC):** hydrocortisone (TDS – higher dose in morning)
   → **Mineralocorticoid (MC):** fludrocortisone (monitor BP, oedema, U&Es, renin)

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[4] NICE (2020) CKS: *Addison's disease*

## Congenital adrenal hyperplasia

→ ***auto-recessive enzyme deficiency for steroid synthesis***
→ ***21-hydroxylase*** *= most common deficiency*

**↓ mineralocorticoids & ↓ glucocorticoids**
**↑ androgens**

**Results in:**

1. ↓ cortisol & ↓ aldosterone synthesis (in 80% 21-hydroxylase deficiency)
2. Backlog of precursors (progesterone)
3. Increased conversion to androstenedione = ↑ **sex hormones**

### CLINICAL PRESENTATION:

| | **Classical (salt-losing)** | **Non-classical (non-salt losing)** |
|---|---|---|
| **Age of presentation** | First few days of life | Teenagers / young adults |
| **Symptoms** | **Hyperandrogenism:**<br>ambiguous genitalia<br>**Similar to Addison's crisis**<br>• hypotension & dehydration (LOC)<br>• N&V, FTT, poor feeding<br>**↑ $K^+$ & ↓ $Na^+$, ↓ Glucose** | **Hyperandrogenism:**<br>early puberty, menstrual irregularity, hirsutism<br>**Enough enzyme to avoid salt loss** |
| **Investigations** | **1.** Steroid hormones & precursor levels | **2.** U&Es & glucose |
| **Management** | Lifelong MC & GC replacement (hydrocortisone & fludrocortisone) | **Minimum dose** GC replacement (enough to ↓ androgen production but not enough to cause Cushing's) |

## Conn's syndrome

→ *1° hyperaldosteronism resulting in $Na^+$ & $H_2O$ retention and $K^+$ loss*

**↑ ALDOSTERONE**
–ve feedback on renin–angiotensin system =
**↓ renin**

NB 2° hyperaldosteronism = high aldosterone & high renin
e.g. chronic liver disease, heart failure, renal artery stenosis

### CAUSES:

→ **Adrenal adenoma (1/3)**
→ Unilateral adrenal hypersecretion without adenoma (1/3)
→ Bilateral adrenal hyperplasia (1/3)

### CLINICAL PRESENTATION:

usually asymptomatic

→ Hypertension (headaches if severe)
→ Hypokalaemia (possible weakness, cramps, tetany)

### INVESTIGATIONS:

1. **BP** = high
2. **U&Es** = ↑ $Na^+$ & ↓ $K^+$
3. **Aldosterone:renin ratio** = high (↑ aldosterone : ↓ renin) DIAGNOSTIC
   → ↑ *aldosterone : normal renin = 2° hyperaldosteronism*
   → *Second line testing = saline infusion or captopril challenge*
4. **Adrenal gland imaging:** CT/MRI abdomen
5. **Localisation:** with adrenal venous sampling

*Suspect Conn's in **younger patients** (30–50y) with **hypertension** plus **hypokalaemia***

### MANAGEMENT:

1. **Adrenal adenoma:** gland resection
2. **Bilateral hyperplasia:** medication (as cannot remove both glands)
   - **Spironolactone/eplerenone** (= aldosterone antagonists)

## Phaeochromocytoma

**↑ Adrenaline/NA**

**Syndromes associated with phaeochromocytoma:**

- **MEN-II** – medullary thyroid carcinoma, hyperparathyroidism
- **Von Hippel-Lindau** – renal cell carcinoma, haemangioblastoma, retinal angioma
- **Neurofibromatosis** – café-au-lait spots, neurofibromas

→ *Suspicion of syndrome if* ***bilateral*** *tumours*

→ ***catecholamine-secreting tumour of adrenal medulla*** *(chromaffin cells)*

**CLINICAL PRESENTATION:** hypertension + classic triad

**TRIAD:** headache + tachycardia + sweating (dizzy/palpitations/tremor)

**INVESTIGATIONS:**

1. **24h urinary metanephrines** ×2 OR **plasma metadrenalines**
2. **Adrenal imaging:**
   - CT/MRI
   - octreotide scan if abnormal CT/MRI
3. **Genetic testing:**
   - if suspicious

**MANAGEMENT:**

**Surgery** (pre-op BP control with alpha- & beta-blockers)

## Cushing's syndrome

**↑ CORTISOL**

→ *symptoms of increased glucocorticoids*

**CAUSES:**

**ACTH-dependent**
**↑ ACTH = ↑ cortisol**

**Cushing's disease** (80%)
= ↑ ACTH secretion
**from pituitary**
e.g. adenoma

**Ectopic ACTH**
= ↑ ACTH secretion
**from elsewhere**
e.g. small cell lung cancer

**ACTH-independent**
**↑ Cortisol = ↓ ACTH**

**Exogenous steroids**

**Adrenal tumour**
= secretes cortisol

**Cushing's syndrome** is umbrella term for all causes of raised cortisol

**Cushing's disease** is specific problem of increased ACTH secretion from pituitary

**CLINICAL PRESENTATION: cortisol has some MC activity so may see ↓ $K^+$, ↑ $Na^+$**

| Symptoms | Signs |
|---|---|
| Central obesity (+ diabetic Sx) | Moon face & frontal balding |
| Acne & excess hair | Buffalo hump |
| Thin skin & bruising | Abdominal striae |
| Depression & insomnia | Proximal myopathy |
| Reduced libido | Pathological fractures |
| Amenorrhoea | Hypertension/DM |

**INVESTIGATIONS:**

**Screening tests for Cushing's syndrome:**

1. **Low dose dexamethasone suppression test (1mg)** = measure 8am cortisol
   → dexamethasone = a GC so should cause ↓ ACTH and therefore ↓ **morning cortisol**
   → **no suppression** in morning cortisol = ***Cushing's syndrome***
2. **24h urinary or midnight salivary cortisol:** lost circadian rhythm

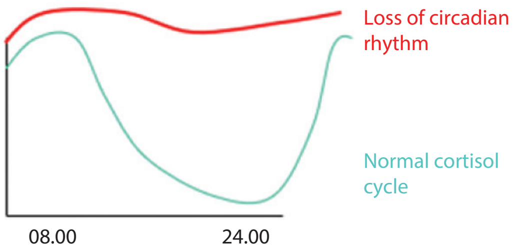


**Fig. 2.6** 24h cortisol levels.

### Determining cause of Cushing's syndrome:

1. **Serum ACTH:** raised in ACTH-dependent causes
2. **CRH stimulation test** – if serum ACTH levels rise >35%, suggests Cushing's disease (<35% suggests ectopic source)
3. **High dose dexamethasone suppression test (2mg QDS for 48h)** = measure 0 & 48h cortisol

   → suppression = Cushing's disease (high dose steroid = enough to suppress pituitary adenoma cells)

   → no suppression = ectopic source (not part of endocrine axis feedback loop, therefore not affected by negative feedback)
4. **Imaging:** MRI (50% pituitary adenomas = too small to detect)
5. **Inferior petrosal sinus sampling** if MRI is normal

Normally do CRH and high dose dexamethasone test in combination

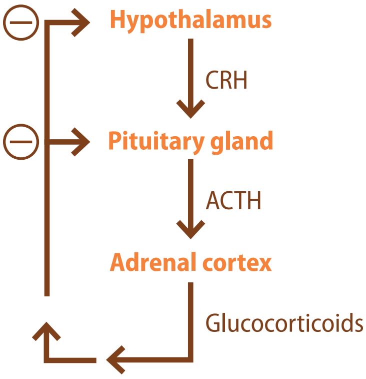

⊖
Hypothalamus
CRH
⊖
Pituitary gland
ACTH
Adrenal cortex
Glucocorticoids

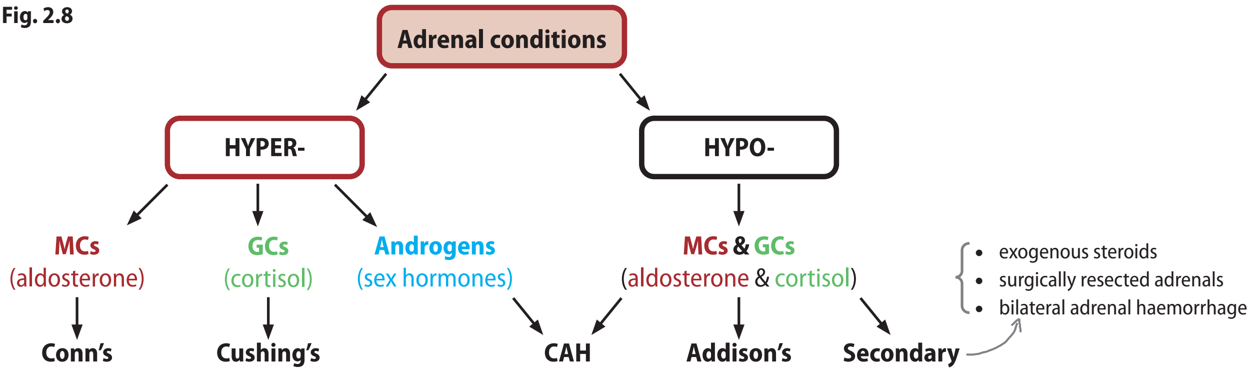
**Fig. 2.7** HPA axis.

## MANAGEMENT:

1. **Cushing's disease:** transsphenoidal resection of pituitary adenoma (± radiotherapy)
2. **Medical therapy** to inhibit cortisol synthesis in adrenal gland
3. **Adrenalectomy** = last resort → will need steroid cover post-op as ↓ endogenous levels

## SUMMARY OF ADRENAL CONDITIONS:

**Fig. 2.8**


Adrenal conditions
HYPER-
HYPO-
MCs
(aldosterone)
GCs
(cortisol)
Androgens
(sex hormones)
MCs & GCs
(aldosterone & cortisol)
Conn's
Cushing's
CAH
Addison's
Secondary
exogenous steroids
surgically resected adrenals
bilateral adrenal haemorrhage

# Thyroid disease

**Signs specific to autoimmune hyperthyroidism:**

- Goitre
- Pre-tibial myxoedema
- Acropachy – swollen hands & clubbing
- **Thyroid eye disease:**
    - exophthalmos – bulging eyes
    - lagophthalmos – cannot close eyes
    - periorbital oedema

**Thyroid function tests (TFTs):**

**Primary:** ↑$T_3$/$T_4$, ↓TSH
**Secondary:** ↑TSH, ↑$T_3$/$T_4$*
**Subclinical:** normal $T_3$/$T_4$, ↓TSH

**Raised TSH & $T_3$/$T_4$ often due to* ***poor compliance*** *or* ***assay interference***

**Medication side-effects**

**Anti-thyroid drugs:**
1. Rash / cholestatic jaundice
2. Agranulocytosis: infection & bleeding risk
→ FBC before Tx and **monitor closely**

**Radioactive iodine:**
= risk of hypothyroidism

**Safety-netting/follow-up:**

1. **Signs of agranulocytosis:**
= sore throat, mouth ulcer, bruising

2. **Signs of thyroid crisis:**
= severe ↑ HR & temp, confusion
(triggers: illness, stress, surgery, V&D)

3. **Signs of tracheal compression**
= SOB/stridor/dysphagia

## Hyperthyroidism

*→ raised circulating thyroid hormones $T_3$ & $T_4$ (= thyrotoxicosis)*

F > M

### GENERAL SYMPTOMS:

- Anxiety, irritability, insomnia
- Fatigue/weakness
- Hot/sweaty, tremor/palpitations
- Menorrhagia/diarrhoea
- ↓Weight (but ↑appetite)

### GENERAL SIGNS:

- ↑HR/arrhythmia
- ↑SBP
- Hyperreflexia
- ± Goitre

### CAUSES:

1. **Autoimmune / Graves' disease (70%) RF:** P/FHx autoimmune disorders
    - Autoimmune stimulation of thyroid follicular cells
    - IgG autoantibodies (TRAb & TPOAb)
2. **Toxic multinodular goitre (15%) RF:** elderly / iodine deficient
    - $T_3$/$T_4$ secreting nodules irresponsive to –ve feedback
3. **Solitary toxic adenoma (5%)**
    - Benign $T_3$/$T_4$ secreting nodule (avg. 3mm) irresponsive to –ve feedback
4. **Drug-induced:** iodine, amiodarone, lithium
5. **Secondary causes (rare):** TSH-secreting pituitary adenoma, pregnancy

### INVESTIGATIONS:

- **Hx & examination** – obs, reflexes etc.
- **TFTs** (TSH, free $T_3$ & $T_4$)
- **Autoantibodies** (TRAb, TPOAb)
- **Technetium uptake scan**
    - if no autoantibodies
    - patchy uptake in nodules
    - diffuse uptake in Graves'
- **Assess eye disease (Graves')**
    - visual fields / eye movements
    - CT/MRI of orbit

### MANAGEMENT:

HYPERthyroidism Mx needs specialist input

1. **Symptomatic relief:** beta-blockers e.g. propranolol, bisoprolol
2. **Anti-thyroid drugs:** carbimazole/propylthiouracil
    - Titration regimen: high dose then titrate to maintain euthyroid
    - Block & replace*: maintain high dose + levothyroxine replacement
3. **Radioactive iodine ($^{131}$I)**

    *Not suitable if pregnant

    - Contraindications: pregnancy/lactation, <16y
4. **Thyroidectomy:** if compression Sx, malignant nodule, Tx-resistant
    - **Post-op complications:** hypothyroidism, hypocalcaemia, vocal cord paresis / hoarseness

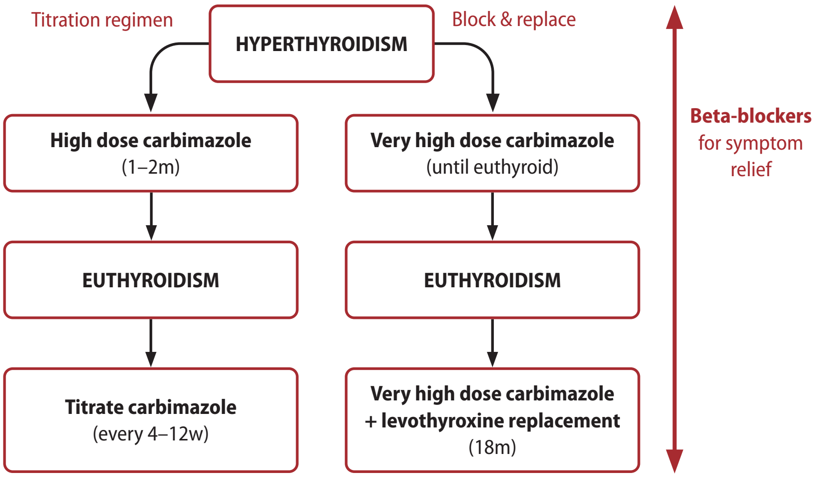

Titration regimen
HYPERTHYROIDISM
Block & replace
High dose carbimazole (1–2m)
Very high dose carbimazole (until euthyroid)
EUTHYROIDISM
EUTHYROIDISM
Titrate carbimazole (every 4–12w)
Very high dose carbimazole + levothyroxine replacement (18m)
Beta-blockers for symptom relief

Fig. 2.9

## Hypothyroidism

→ *low circulating thyroid hormones $T_3$ & $T_4$ (= thyrotoxicosis)*

F > M

**GENERAL SYMPTOMS:**

- Depression/psychosis
- Slowed intellectual activity
- Fatigue/weakness
- Cold
- Amenorrhoea/constipation
- ↑ Weight
- ↓ Libido

**GENERAL SIGNS:**

- ↓ HR
- Anaemia
- Hyporeflexia
- Dry skin
- Hair loss (outer 1/3 eyebrow)

± non-pitting oedema
± goitre

**CAUSES:**

*Most common cause in UK

1. **Autoimmune /Hashimoto's thyroiditis*: RF:** autoimmune disorders, 60–70s
   - Autoantibodies against TPO (TPOAb)
   - Gradual gland destruction: lymphocyte infiltration & fibrosis → **may have goitre**
   - Initial hyperthyroid state: damage releases $T_3$/$T_4$
2. **Primary atrophic thyroiditis:**
   - Autoimmune gland destruction
   - Extensive lymphocyte infiltrate atrophies gland → **no goitre**
3. **Previous hyperthyroidism treatment**
   - Post thyroidectomy or radioactive iodine
4. **Iodine deficiency** (most common cause worldwide)
5. **Drug-induced:** amiodarone, lithium, carbimazole, excess iodine
6. **Congenital:** absence/underdevelopment of thyroid gland or enzyme deficiency
7. **Postpartum thyroiditis:** development within 1y of giving birth (lasts 4–6m)
8. **Secondary causes (very rare):** hypopituitarism

**INVESTIGATIONS:**

- **Hx & examination** – observations, reflexes, skin changes
- **TFTs** (TSH, free $T_3$ & $T_4$)
- **FBC** – for anaemia
- **Autoantibodies** (TPOAb)

**Starting doses of levothyroxine:**
**18–49y:** 50–100mcg
**>50y or CVD:** 25–50mcg

**MANAGEMENT[5]:**

**Thyroid hormone replacement therapy:** levothyroxine ($T_4$)

- Low dose & titrate up until desired TSH levels reached
  - review dose monthly initially, then every 3–4m
  - review dose annually once stable
- **Lifelong therapy**

Patients with goitre, suspected malignancy, cardiac disease, treatment resistant or planning a pregnancy need referral to endocrinologist in 2° care

**DDx of hypothyroidism:**

- Diabetes mellitus
- Adrenal insufficiency
- Coeliac disease
- Anxiety/depression
- Dementia

**Thyroid function tests (TFTs):**

**Primary:** ↓ $T_3$/$T_4$, ↑ TSH
**Secondary:** ↓ TSH, ↓ $T_3$/$T_4$
**Subclinical:** ↑ TSH but $T_3$/$T_4$ normal
**Sick euthyroid syndrome:** temporary ↓ TSH during illness

**Complications:**

- CVD/stroke
- Dyslipidaemia
- Heart failure
- **Myxoedema coma***

*hypothermia, coma, seizures (precipitated by another problem e.g. sepsis, stroke, HF)

---

[5] NICE (2019) CKS: *Thyroid disease*

## Goitres

### ASSESSMENT:

1. **Hx** – symptoms of compression, speed of growth, PHx radiation, FHx
2. **Exam** – assess size/consistency/distribution
3. **Thyroid gland function** – TFTs
4. **USS ± aspirate** – size, cystic vs. solid **DIAGNOSTIC**
5. **Complications:** pressure on trachea (SOB) / oesophagus (dysphagia) / voice change (recurrent laryngeal nerve) / thoracic inlet obstruction*

*compression of jugular vein & aorta
= causes Pemberton's sign (facial flush if raise arms)

### MANAGEMENT:

1. **No symptoms, euthyroid & benign:** no treatment
2. **If hypothyroidism:** thyroxine
3. **If autoimmune disease:** antithyroid drugs / radioiodine / surgery
4. **Surgery:** if large / pressure symptoms / cosmetic / malignant

**DDx of goitres:**
1. **Physiological**
2. **Autoimmune:** Graves', Hashimoto's
3. **Multinodular:** iodine deficiency
4. **Solitary nodule:** cyst, adenoma, carcinoma

**Goitre classification:**

**0** – can't see/feel
**1** – palpable not visible
**2** – visible in normal position

## Thyroid cancer

### CLINICAL PRESENTATION:

- **90% = thyroid nodules/goitre → usually asymptomatic**
- Hoarseness/dysphagia
- Thyroid dysfunction (rare)

### INVESTIGATIONS:

1. Hx & examination – PHx radiation, thyroid disease, FHx
2. USS
3. FNA/biopsy – for cytology GOLD STANDARD Ix
4. Further imaging if indicated: PET / technetium scan
5. Tumour markers **if cancer confirmed** – thyroglobulin (+ calcitonin if medullary cancer)

**Epidemiology of thyroid cancer**
- F:M = 3:1
- peak onset 30–50y
- RF = Asian

**Prognostic factors:**
- Size of tumour
- Local invasion & distant mets
- Age of patient

### TYPES OF THYROID CANCER:

| | Papillary (75%) | Follicular (20%) | Medullary (5%) | Anaplastic (rare) |
|---|---|---|---|---|
| **Origin** | Follicular cells | Follicular cells | C cells | Any cells |
| **Features** | • Slow growth<br>• LN metastases | • Slow growth<br>• Bone/lung mets | • May release calcitonin | • Fast growth<br>• Very invasive<br>• Distal mets |
| **Who?** | 30–50y females | | 1. Sporadic = older patients<br>2. Part of **MEN-II syndrome** = younger | Elderly (M:F = 2:1) |
| **Prognosis** | **Good:** 80% 10y | **Good:** 65% 10y | **Poor** | **Very poor:** palliative |

**Well differentiated** (Papillary, Follicular) **Poorly differentiated** (Medullary, Anaplastic)

***MEN-II syndrome:***
*medullary thyroid carcinoma + phaeochromocytoma + hyperparathyroidism*

### MANAGEMENT:

1. **Total thyroidectomy**
2. TSH suppression – thyroxine (may need for years)
3. Follow-up – radioactive iodine (RAI) scan & tumour marker monitoring
4. *Therapeutic dose of RAI if residual disease*

# Parathyroid disease

## Calcium homeostasis

1. **PTH secretion if:**
    - Low serum Ca
    - (or low vit D / high $PO_4$)
2. **PTH increases serum Ca by:**
    - ↑ Osteoclastic bone resorption
    - ↑ Ca reabsorption in kidney
    - Activating vit D in kidney

**Vitamin D function:** activated in kidney
1. Increase Ca absorption in gut
2. Increase osteoblastic bone formation

**Calcitonin function:** secreted by parafollicular (C/Chief) cells of thyroid if **high** serum Ca

## Hypercalcaemia (Ca >2.6mmol/L)

### CAUSES:

| | |
|---|---|
| **↑ PTH secretion**<br>(*high* PTH = ↑ Ca) | → 1° Hyperparathyroidism **e.g. adenoma**<br>→ 2° Hyperparathyroidism e.g. long-term CKD, vit D deficient<br>→ 3° Hyperparathyroidism e.g. renal failure |
| **Malignancy**<br>(↑ Ca = *low* PTH) | → Breast, bronchus, kidney, prostate<br>→ Multiple myeloma |
| **Vit D (calcitriol) excess** | → Tablets<br>→ Granulomatous disease e.g. sarcoid, TB |
| **Hereditary**<br>(↑ serum Ca, ↓ urine Ca) | → Hypocalciuric hypercalcaemia |
| **Endocrine** | → Addison's, thyrotoxicosis |
| **Medication** | → Thiazide diuretics, lithium |

**90% cases**

### SYMPTOMS:

1. **Bones:**
    - fractures/pain
    - muscle weakness
2. **Stones:**
    - renal stones
    - AKI/CKD
3. **Abdo groans:**
    - abdo pain, N&V
    - constipation
    - pancreatitis
4. **Psychic moans:**
    - depression
    - confusion
    - ↓ consciousness

### INVESTIGATIONS:

1. **Corrected serum Ca, PTH & vit D, $PO_4$ + U&Es**
2. **24h urinary Ca:** r/o familial hypercalcaemia
3. **Imaging:**
    - DEXA scan – bone density
    - Renal USS/XR – renal stones
    - Localisation study – USS neck + sestamibi scan

**40% Ca is bound to albumin = inactive**
→ If low albumin, Ca will appear low in tests
→ **Adjust for albumin** (shows ionised calcium)

### MANAGEMENT OF ACUTE SEVERE HYPERCALCAEMIA[6]: >3mmol/L

→ triggers osmotic diuresis → DEHYDRATION

1. **Rehydration:** 0.9% saline (4–6L over 24h)
2. **Loop diuretics:** increase calcium excretion (only if fluid overload)
3. **IV bisphosphonates** if malignant (e.g. zoledronate → take 3–5d to work)
4. **IV calcitonin:** if aggressive presentation

Check serum Ca, U&Es, hydration status

**Causes of vitamin D deficiency:**
- Little sunlight
- Malabsorption
- Liver/kidney disease

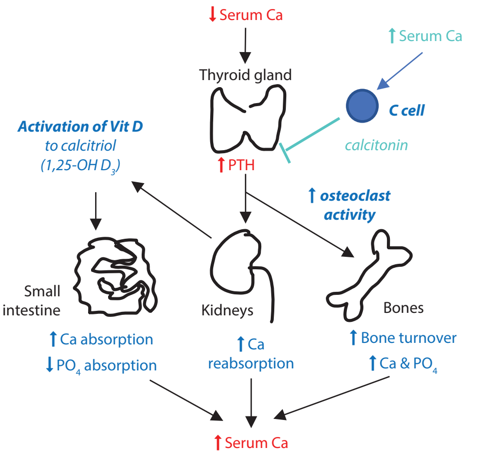


**Fig. 2.10** Calcium homeostasis.

Chronic 2° hyperparathyroidism causes permanent gland activation = 3° hyperparathyroidism

**Primary hyperparathyroidism**

→ peak age 60y (F>M)
→ **Causes:**
- Single adenoma – **most common**
- Multiple adenomas – consider MEN-I syndrome

→ **Investigations:** USS, isotope scan
→ **Treatment:** resection/parathyroidectomy **or** calcium mimetics e.g. cinacalcet

**Interpreting blood tests**

| | PTH | ALP |
|---|---|---|
| **1° HPT** | High | High |
| **Malignancy** | Low | High |

**If worried about cancer:** CT CAP, myeloma screen, PSA (to find primary)

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[6] Society For Endocrinology Endocrine Emergency Guidance – Emergency management of acute hypercalcaemia in adult patients (2016)

## **Hypocalcaemia** (Ca <2.2mmol/L)

### CAUSES:

| | |
|---|---|
| **↓ PTH secretion**<br>(*low* PTH = ↓ Ca) | → **1° Hypoparathyroidism**<br>e.g. post-thyroidectomy, autoimmune<br>→ **Hypomagnesaemia** = impairs PTH secretion<br>e.g. commonly caused by PPIs |
| **PTH resistance**<br>(*high* PTH but ↓ Ca) | → **Pseudohypoparathyroidism***<br>= PTH receptor defect |
| **Hyperphosphataemia**<br>($PO_4$ binds to free Ca) | → CKD, rhabdomyolysis, phosphate therapy |
| **Vit D deficiency**<br>(*high* PTH but ↓ Ca) | → Malabsorption = Crohn's / coeliac<br>→ Inadequate diet / sun exposure |
| **Acute pancreatitis** | → Sequestration of calcium into tissues |
| **Medication** | → Calcitonin or bisphosphonates |

*Inherited disorder only affecting females

Serum calcium appears ARTIFICIALLY LOW if do not adjust for albumin

### SYMPTOMS:

→ **Peripheral paraesthesia:**
- Numbness/tingling periorally/fingers

→ **Muscle cramps/spasms/tetany:**
- Trousseau's sign = claw hand with BP cuff
- Chvostek's sign = facial nerve twitching when tapped

→ **Fits/seizures/arrhythmia: if <1.9mmol/L**

→ **Long-term complications:** depression, cataracts, bone fractures

### INVESTIGATIONS:

1. **Corrected serum Ca, PTH & vit D, $PO_4$**
2. **ECG:** prolonged QT interval

### MANAGEMENT[7]:

**Acute mild hypocalcaemia (>1.9mmol/L):**

→ **Oral calcium supplements:** e.g. Adcal 3, Calcichew Forte 2, Sandocal

**Acute severe hypocalcaemia (≤1.9mmol/L):**

→ **Initial IV bolus:** 10–20ml 10% calcium gluconate (in 50–100ml 5% dextrose over 10min with ECG monitoring)

→ **Followed by IV infusion:** 100ml 10% calcium gluconate (in 1L normal saline or 5% dextrose over 5–10h)

→ **2nd line:** oral calcium carbonate (1g TDS) + PO/IM alfacalcidol (1–2mcg)

**Chronic hypocalcaemia:** treat cause & monitor

→ **Hypoparathyroidism:** vit D supplements (calcidiol/calcitriol/alfacalcidol) ± oral calcium supplements

### MEN syndrome

| MEN-I | MEN-IIa | MEN-IIb |
|---|---|---|
| **1.** Pituitary adenoma<br>**2.** Parathyroid hyperplasia<br>**3.** Pancreatic neuroendocrine tumour | **1.** Parathyroid hyperplasia<br>**2.** Medullary thyroid carcinoma<br>**3.** Phaeochromocytoma | **1.** Mucosal neuromas<br>**2.** Marfanoid body habitus<br>**3.** Medullary thyroid carcinoma<br>**4.** Phaeochromocytoma |

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[7] Society For Endocrinology (2016) *Endocrine Emergency Guidance – Emergency management of acute hypocalcaemia in adult patients*

# Sodium imbalance

## **Hypernatraemia** (Na >146mmol/L)

### SYMPTOMS:

- Headache
- N&V
- Lethargy/irritability/confusion
- Seizures/nystagmus
- Loss of consciousness / coma

### INVESTIGATIONS/ WORK-UP:

- Fluid status
- Obs – especially BP & neuro Sx
- Medication review
- **Bloods:** U&Es, glucose
- **Osmolality:** plasma & urine
  - **Urine osm >750** suggests lack of water
  - **Urine osm <750 / lower than plasma** suggests diabetes insipidus

### CAUSES:

1. **Low fluid intake:** RFs = age >65y, dementia, residential care
2. **Diabetes insipidus:** central or nephrogenic
3. **Hyperosmotic hyperglycaemic state (HHS):** osmotic diuresis = water loss
4. **Other:** endocrine disorders e.g. Conn's, Cushing's

### MANAGEMENT:

- **Rehydrate*** – orally, if possible, otherwise with IV fluids
- Daily U&Es

*Reducing plasma Na too quickly can cause cerebral oedema

## **Hyponatraemia** (Na <133mmol/L)

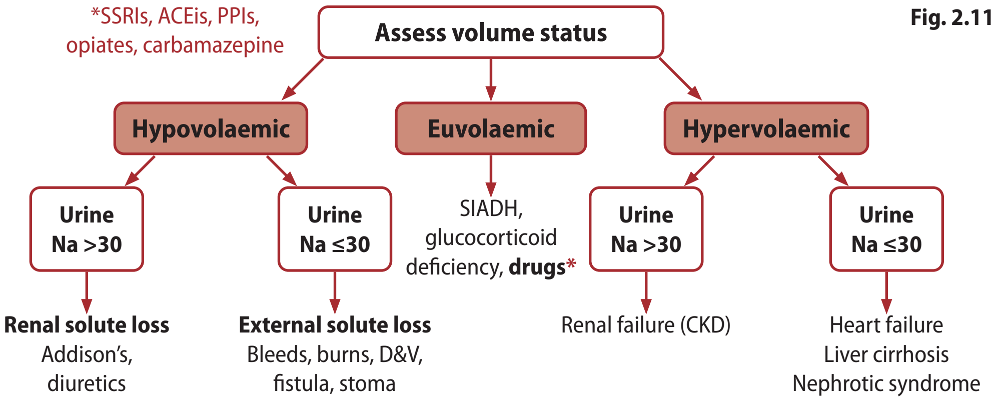
### SYMPTOMS:

- Headache
- N&V
- Lethargy/irritability/confusion
- Seizures
- Loss of consciousness / coma

### INVESTIGATIONS/ WORK-UP:

- **Fluid status**
- **Obs** – especially BP & neuro Sx
- **Medication review**
- **Bloods:** U&Es, glucose, LFT, total protein, lipids, Ca, TFT, cortisol
- **Osmolality:** plasma & urine

### DETERMINING THE CAUSE:



*SSRIs, ACEis, PPIs, opiates, carbamazepine
Assess volume status
Hypovolaemic
Euvolaemic
Hypervolaemic
Urine Na >30
Urine Na ≤30
SIADH, glucocorticoid deficiency, drugs*
Urine Na >30
Urine Na ≤30
Renal solute loss
Addison's, diuretics
External solute loss
Bleeds, burns, D&V, fistula, stoma
Renal failure (CKD)
Heart failure
Liver cirrhosis
Nephrotic syndrome

**Fig. 2.11**

### MANAGEMENT:

**Hypovolaemic:** IV fluids (0.9% NaCl)
**Hypervolaemic:** consider fluid restriction + treat underlying cause
**Neurological symptoms present:** IV hypertonic saline

→ **PLUS** review/stop causative medications

**Hypernatraemia is usually due to water deficiency/loss** rather than excess sodium intake

**Indications for urgent admission:**

- Na >155 (or >146 & cannot drink)
- Neurological disturbance
- Presence of HHS

**Diabetes insipidus**

**Central:** lack of ADH secretion e.g. head injury, pituitary tumour, pituitary surgery
**Nephrogenic:** renal resistance to ADH e.g. 2° to lithium Tx / renal disease

Sx: polyuria & polydipsia

Ix: high plasma osm, low urine osm

Mx:
**Central** = desmopressin
**Nephrogenic** = ↓salt diet, hydration, amiloride

*Other Ix depend on cause:*
*CXR, BNP, brain MRI / CT head*

**Pseudohyponatraemia:**
Na levels appear low when very elevated lipid or protein levels

**Caution:** do not correct hyponatraemia too rapidly
→ risk of **cerebral pontine myelinosis (CPM)**

# 03

# GASTROENTEROLOGY

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## ABBREVIATIONS

**Al** – Aluminium
**5-ASA** – 5-aminosalicylic acid
**BMI** – Body mass index
**BMR** – Basal metabolic rate
**Ca** – Calcium
**CBT** – Cognitive behavioural therapy
**CCB** – Calcium channel blocker
**CHO** – Carbohydrate
**Cr** – Creatinine
**CRC** – Colorectal carcinoma
**D&V** – Diarrhoea and vomiting
**DEXA** – Dual-energy X-ray absorptiometry
**ERCP** – Endoscopic retrograde cholangiopancreatography
**FB** – Foreign body
**FTT** – Failure to thrive
**GI** – Gastrointestinal
**GIST** – Gastrointestinal stromal tumour
**GOJ** – Gastro-oesophageal junction
**GORD** – Gastro-oesophageal reflux disease
**GTN** – Glyceryl trinitrate
**$H_2RA$** – $H_2$ receptor antagonist
**HNPCC** – Hereditary non-polyposis colorectal cancer
**IAP** – Intra-abdominal pressure
**IBD** – Inflammatory bowel disease
**IBS** – Irritable bowel syndrome
**IDA** – Iron-deficiency anaemia
**LN** – Lymph node
**Mg** – Magnesium
**MG** – Myasthenia gravis
**MS** – Multiple sclerosis
**MUAC** – Mid-upper arm circumference
**NAFLD** – Non-alcoholic fatty liver disease
**NBM** – Nil by mouth
**OA** – Osteoarthritis
**OGD** – Oesophago-gastroduodenoscopy
**PAL** – Physical activity level
**PBC** – Primary biliary cirrhosis
**PET** – Positron emission tomography
**$PO_4$** – Phosphate
**PPI** – Proton pump inhibitor
**PSC** – Primary sclerosing cholangitis
**PUD** – Peptic ulcer disease
**SI** – Small intestine
**SSRI** – Selective serotonin reuptake inhibitor
**TCA** – Tricyclic antidepressant
**TIPS** – Transjugular intrahepatic portosystemic shunt
**tTG** – Tissue transglutaminase
**UC** – Ulcerative colitis

# Clinical nutrition

**Daily fluid needs:** 30–35ml/kg (2L/day)
+ 500ml per degree pyrexia

**Energy composition**

**60% CHO** (4kcal/g)
**30% Fat** (9kcal/g)
**15% Protein** (4kcal/g) = 0.8–1.5g/kg/d

**Daily electrolyte requirements:**
**Na & K:** 1mmol/kg
**Ca, Mg, $PO_4$:** 0.1mmol/kg

| Waist circum. in obesity | Normal fold thickness |
|---|---|
| Women >88cm | Women =30mm |
| Men >102cm | Men =20mm |

Albumin = poor measure of nutritional status as drops in inflammation

BMR = Basal metabolic rate
PAL = Physical activity level

**DAILY ENERGY EXPENDITURE = BMR x Stress factor x PAL**

**BMR** = 1.3kcal/kg/h
= 25–30kcal/kg/d
= 2000–2500kcal/d

**Surgery** = 1.2
**Wound/infection** = 1.5
**Severe burn** = 2.0

**Bed-bound** = 1.2
**Light activity** = 1.5
**Moderate activity** = 1.8
**High activity** = 2.2

## Assessing nutritional status

→ *body composition, function & growth*

1. **History**
   - Dietary details – what, when, how much? any changes?
   - Noted weight or appetite loss/change
   - GI symptoms – N&V, diarrhoea, constipation, pain
   - Systemic symptoms – fatigue, neurological, oedema
   - *Growth / FTT – in children*
2. **Calculate MUST score (STAMP chart in children)**
3. **Anthropometry**
   - Waist circumference & waist:hip ratio
   - Height, weight & BMI ($w/h^2$)
   - *MUAC & growth charts – in children*
   - Skin fold thickness – subcutaneous fat
   - Grip strength
   - Bioelectric impedance/DEXA – body composition
4. **Blood tests – for specific deficiencies**
   - Hb, iron studies, B12 & folate – anaemia
   - Urea & Cr – protein turnover / dehydration
   - Core electrolytes – Na, K, Ca, Mg, $PO_4$
   - ± Fat-soluble vitamins – Vitamin A, D, E (measure **clotting for Vitamin K**)

## Nutritional support

| Enteral tube | Indications | Complications/disadvantages |
|---|---|---|
| **NG tube** (nasogastric) | • Poor dentition/oral pathology<br>• Inflamed mucosa (chemo/candidiasis)<br>• Swallow problems (stroke/MND)<br>• Aspiration risk (↓ GCS, laryngeal nerve palsy)<br>• Obstructing stricture (cancer, radiotherapy)<br>• *Aspiration of stomach contents* (*bowel obstruction*) | • **Displaced by coughing/vomiting** (check position via CXR)<br>• **Misplaced tube (into lung)** (pneumothorax, pneumonia, death)<br>**Remove if:**<br>1. Displaced<br>2. Recovered<br>3. >6w |
| **NJ tube** (nasojejunal) | • Severe vomiting (e.g. hyperemesis gravidarum)<br>• Gastric cancer, causing outlet obstruction<br>• Gastroparesis (in diabetes)<br>• Pancreatic cancer, causing duodenal obstruction | • **Need more complex, pre-digested formula**<br>• **Require radiological/endoscopic placement**<br>• **Less likely to displace** (can send patients home with them) |
| **PEG/RIG tube** (percutaneous) | • More comfortable on pharynx/oesophagus<br>• Avoid nasal erosion<br>• **If longer term nutrition needed (>4m)** e.g. stroke with poor swallow, throat cancer | • **Cannot remove if been inserted <6w** (tube tract not yet healed so complications)<br>• **Needs radiological/endoscopic placement**<br>• **Stoma complications** |

**STEPWISE APPROACH to nutritional support:**
DIET ADVICE
↓
FOOD FORTIFICATION
↓
ORAL SUPPLEMENTS
↓
PAR-/ENTERAL FEEDING

**PARENTERAL FEEDING:** →

**Indications:**

- Shortened bowel with **high output stoma**
- Dysfunctional bowel e.g. ileus, colitis
- Contraindicated for enteral feeding e.g. no access
- ***Need to replace trace elements:*** *vitamins & minerals*

**Complications**
→ Infection of access site
→ Venous thrombosis

Must monitor FBC, U&Es, LFT, bone profile & magnesium

**Metabolic complications:**
→ Higher level of bone turnover (causes osteoporosis)
→ Associated **liver dysfunction** (check LFTs)
→ Risk of over- or under-hydration

# Gastro-oesophageal reflux disease

**20% adults experience heartburn**
**M:F = 3:1**

## Symptoms

- Retrosternal pain
  - **worse lying/bending** down & with hot liquid / alcohol
  - **relieved by antacids**
- Regurgitation/waterbrash – sudden filling of mouth with saliva
- Odynophagia
- Atypical chest pain
- Nocturnal wheeze/cough
- Tooth decay

## Causes

1. **Anatomical:** sphincter dysfunction/ hiatus hernia (sliding/rolling)
   - Sliding: GOJ + part of fundus above diaphragm
   - Rolling: only part of fundus above diaphragm (↑ risk strangulation)
2. **Physiological:**
   - Smoking/alcohol
   - Spicy/fatty food, large meals late at night
   - Drugs (anticholinergics, nitrates, TCAs, CCBs)
   - ↑ IAP (pregnancy/obesity)

## Investigations

- **History** – diagnosis made on clinical symptoms
- **FBC** – check for anaemia
- **Ambulatory 24h pH testing** – press record when getting symptoms → use to calculate **DeMeester score**
- **Barium swallow** – identify anatomical causes of GORD
- **High resolution manometry (HRM)** – measures oesophageal pressure / sphincter function
- **OGD (oesophago-gastroduodenoscopy)** – done if ALARM symptoms via **urgent 2ww referral**

## Initial management[1]

1. **LIFESTYLE**
   - Weight loss, ↓ smoking & alcohol, ↓ stress
   - Smaller meals >3h before bed, avoid spicy food
   - Raise head of bed / sleep in a more upright position
   - **Medication review:** anticholinergics, nitrates, TCAs, CCBs, NSAIDs
2. **MEDICAL TREATMENT:**
   - Antacid/alginates
   - Full dose PPI (4–8w) e.g. omeprazole 20mg OD
   - $H_2$ receptor antagonist (2nd line / PPI contraindicated)
3. **SURGICAL TREATMENT:** restore anatomical position of stomach & GOJ, repair herniae & recreate antireflux valve
   - NISSEN FUNDOPLICATION = 360° wrap of fundus around lower GOJ

**Indications for surgery** → Failure of medical Tx → Do not want lifelong PPI
→ Extra-oesophageal Sx (wheeze, hoarse, cough, chest pain, aspiration)
**+ Good evidence of reflux on pH/manometry testing**

### Dyspepsia (indigestion):

**Symptoms:** abdo pain, bloating, N&V, heartburn, food/acid regurgitation

**DDx:**

- GORD
- Gastric cancer
- PUD (gastric/ duodenal)
- Oesophageal cancer

**Complications of GORD:**
**In adults:** oesophagitis/ulcers → Barrett's → adenocarcinoma
**In children:** aspiration pneumonia, frequent otitis media
**In infants:** feeding difficulties, ↓ growth, distressed behaviour

### Red flags / alarm symptoms

**A**naemia (Fe deficient)
**L**ost weight
**A**norexia
**R**ecent onset & progressive
**M**elaena or haematemesis
**S**wallowing difficulties (dysphagia)
**55**y or older (+ one of above)

*Biopsy assessed with Prague criteria based on height & circumference of epithelium affected

### Barrett's oesophagus:

*stratified epithelium → columnar*

**Sx:** similar to GORD or asymptomatic

**Ix:** same as for GORD **PLUS BIOPSY***

**Mx:** treat GORD

- **No dysplasia:** repeat OGD in 2–5y
- **Low grade:** repeat 6m + ablation
- **High grade:** repeat OGD + resection

↳ **Risk of adenocarcinoma in 1y:**
Low grade dysplasia: 0.7%
High grade dysplasia: 7%

NB a side-effect of surgery = dysphagia

---

[1] NICE (2014, updated 2019) *Gastro-oesophageal reflux disease and dyspepsia in adults* [CG184]

# Peptic ulcer disease

| **Duodenal** – near pylorus | **Gastric** – lesser curve |
|---|---|
| • **pain relief on eating**/milk<br>• no anorexia/vomiting<br>90% caused by *H. pylori* | • **pain worse on eating**<br>• anorexia & vomiting |

**ZES – rare condition where tumours (gastrinomas) of the pancreas & duodenum secrete gastrin, causing excess stomach acid production*

### Complications of PUD

**Acute bleed**
- endoscopic coagulation/clipping
- interventional radiology (embolisation)

**Perforation**
- urgent surgical repair (Graham patch)

**Stricture**
- endoscopic dilation & stenting
- OR bypass via gastrojejunostomy

### *H. pylori* test & treat

**Test:** $^{13}$C urease breath test or *H. pylori* faecal antigen

**Treat:** triple therapy = PPI + 2 × ABX (metronidazole, amoxicillin, clarithromycin)

### MEDICATIONS:

1. **Antacids:** Na, Mg, Al salts
    - react with acid to ↑ pH
2. **Alginates:** Gaviscon Advance
    - react with acid to form raft
3. **$H_2$RAs:** famotidine/cimetidine
    - block histamine = ↓ $H^+$ release

      *SE: diarrhoea, rash*
4. **PPIs:** omeprazole, lansoprazole
    - irreversible PPI

      *SE: hyponatraemia, **osteoporosis**, CKD, hypomagnesaemia, C. difficile*

## Causes

- *H. pylori* – causes **PUD** or **gastric cancer**
- NSAIDs (+ steroids & SSRIs)
- Zollinger–Ellison syndrome*
- Smoking/caffeine

## Symptoms

- Dyspepsia – retrosternal heartburn
- Burning epigastric pain – related to food/hunger
- ± Haematemesis/melaena

## Investigations

**<55y & no ALARM Sx:** clinical Dx

**>55y or ALARM Sx:** 2ww for endoscopy

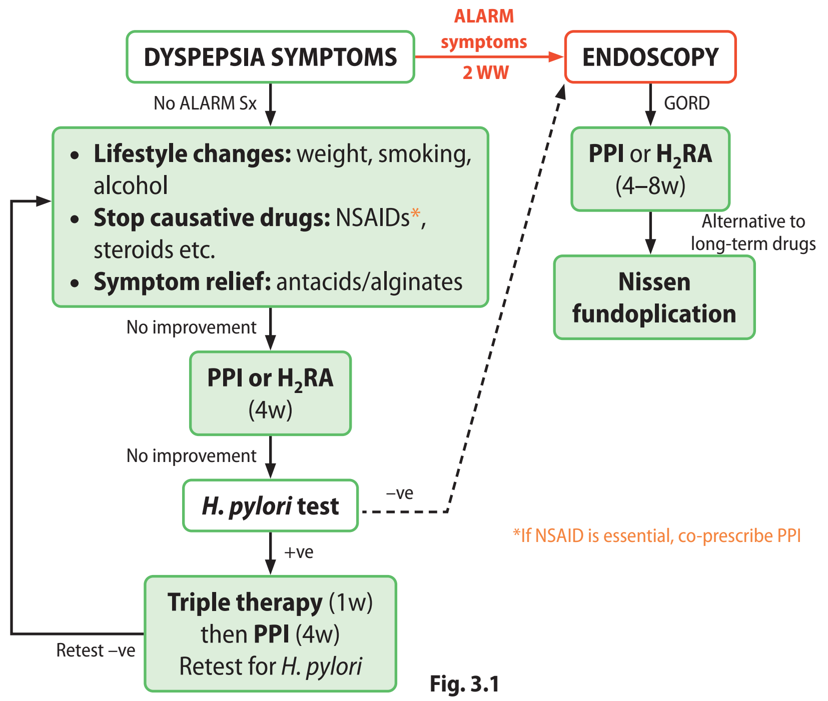
## Management[2]

1. **Lifestyle:** weight loss, ↓ smoking & alcohol, ↓ stress, smaller meals + **medication review** (stop NSAIDs if possible)
2. **Medical therapy:**

→ **1st line:** full dose PPI (4–8w) e.g. omeprazole 20mg OD

→ **2nd line:** *H. pylori* 'test & treat' = triple therapy **(7d)** then PPI

## Summary of dyspepsia management


DYSPEPSIA SYMPTOMS
ALARM symptoms
2 WW
ENDOSCOPY
GORD
PPI or $H_2$RA (4–8w)
Alternative to long-term drugs
Nissen fundoplication
No ALARM Sx
Lifestyle changes: weight, smoking, alcohol
Stop causative drugs: NSAIDs*, steroids etc.
Symptom relief: antacids/alginates
No improvement
PPI or $H_2$RA (4w)
No improvement
H. pylori test
–ve
+ve
Triple therapy (1w) then PPI (4w) Retest for H. pylori
Retest –ve

*If NSAID is essential, co-prescribe PPI

**Fig. 3.1**

---

[2] NICE (2014, updated 2019) *Gastro-oesophageal reflux disease and dyspepsia in adults* [CG184]

# Gastro-intestinal haemorrhage

****Chronic GI bleed** presents as symptomatic or asymptomatic **microcytic anaemia***

| | Upper GI bleed | Lower GI bleed |
|---|---|---|
| **Causes** | - PUD = most common<br>- Mallory–Weiss tear<br>- Oesophageal varices<br>- Drugs (NSAIDs, aspirin, steroids, anticoagulants)<br>- Oesophagitis/gastritis<br>- Malignancy<br>Endoscopic procedures also ↑ risk GI bleed e.g. ERCP | - Diverticular disease<br>- Ischaemic colitis<br>- Carcinoma<br>- Polyps, ulcers, fistulas, haemorrhoids<br>- Angiodysplasia |
| **Symptoms*** | - Haematemesis (bright red) / coffee-ground vomit<br>- Melaena<br>- Hypovolaemic shock (cold, clammy, ↑ HR)<br>± abdo pain | - Melaena or bright red PR bleed (if RHS colon)<br>- Dehydration<br>± abdo pain |
| **Acute management** | 1. **Resuscitation:** ABCDE – blood transfusion if Hb <80<br>2. **History:** medications / RFs for bleed<br>3. **Localise bleed:** endoscopy ± CT angiogram<br>4. **Continuous monitoring & reassessment:** keep NBM if high risk of rebleed | |
| **Investigations** | **Blatchford score:** indicates timing of endoscopy<br>→ discharge if score = 0<br>→ **urgent upper GI endoscopy** if score >0<br>**Rockall score:** predicts mortality from bleed<br>(can only calculate **after** endoscopy) | **Massive bleed:** CT angiogram<br>**Major bleed:** lower GI endoscopy<br>**Minor bleed:** discharge for outpatient Ix |
| **Definitive management**[3] | 1. **Pharmacological**<br>- **Non-variceal:**<br>→PPI (IV or PO), stop NSAIDs, *H. pylori* eradication<br>- **Variceal:** terlipressin<br>2. **Endoscopic therapy:**<br>- Clip / embolise / variceal bands<br>- Danis stent<br>- Sengstaken–Blakemore / Minnesota tube<br>→ *If bleeding continues:*<br>3. **Interventional radiology** (**embolisation, TIPS**)<br>4. **Surgery** | **Massive bleed:**<br>Endoscopic therapy or interventional radiology<br>→ *If bleeding continues = surgery*<br>**Major bleed**<br>Treat cause if found & discharge if bleeding has stopped |

*TIPS: transjugular intrahepatic portosystemic shunt if uncontrolled variceal bleed*

**Glasgow–Blatchford score – done pre-OGD**

| Risk factors considered |
|---|
| Blood urea nitrogen (BUN) |
| Haemoglobin levels |
| Systolic BP |
| Heart rate |
| Melaena |
| Syncope |
| Hepatic disease |
| Cardiac failure |

**Rockall score – done post-OGD**

| Parameters considered |
|---|
| Age |
| Features of haemodynamic shock<br>e.g. hypotension, tachycardia |
| Coexisting illness<br>e.g. CHF, renal failure, cancer |
| Endoscopic signs<br>e.g. erosive disease, peptic ulcer, visible bleed |

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[3] NICE (2012, updated 2016) *Acute upper gastrointestinal bleeding in over 16s* [CG141]

# Dysphagia

**Oropharyngeal dysphagia:** difficulty **initiating swallowing** ± aspiration/choking = **neurological cause**
**Oesophago-pharyngeal dysphagia:** food 'sticks' **after swallowing** ± regurgitation = **dysmotility, stricture, lesion as cause**

## Causes

| | |
|---|---|
| **Neuromuscular** | MG, MS, stroke, Parkinson's |
| **Oesophageal dysmotility** | Achalasia, oesophageal spasm, scleroderma |
| **Extrinsic pressure** | Goitre, lymph nodes, malignancy (of lung) |
| **Intrinsic lesion** | Foreign body, pharyngeal pouch, benign stricture, malignancy, oesophageal web (Plummer–Vinson syndrome) |

**Plummer–Vinson syndrome:**
→ Triad of **dysphagia + IDA + post-cricoid oesophageal web**
→ Pre-malignant formation of oesophageal webs
→ **Mx:** iron + OGD dilation of webs

## Investigations

- **FBC:** for IDA → 2ww for endoscopy
- **Upper GI endoscopy / OGD** (+ biopsy if suspect malignancy)
- **Barium swallow** (only snapshot)
- **Videofluoroscopy** (shows phases of swallow if suspect oropharyngeal dysphagia)
- **High resolution manometry** (measures oesophageal pressure during swallow)

## Achalasia

Degeneration of the myenteric plexus (due to infection, autoimmune, genes) leads to loss of coordinated peristalsis and inability of lower sphincter to relax

**SYMPTOMS:** **regurgitation**, dysphagia, weight loss

**INVESTIGATIONS:**

- High resolution manometry = **gold standard** (↑ pressure of sphincter + lack of peristalsis)
- Barium swallow ('bird's beak')

**MANAGEMENT:** depends on fitness & preference of patient

1. **Surgical:**
    - Laparoscopic Heller's cardiomyotomy + antireflux fundoplication* = cut made in sphincter
2. **Endoscopic:**
    - Peroral endoscopic myotomy (POEM) = new procedure (endoscopic version of Heller's)
    - Balloon dilation (only temporary relief)
    - Botox injection (only temporary relief)
3. **Pharmacologic:** CCB & GTN (relax sphincter **but** lack of evidence & rarely used)

*Fundoplication to ↓ reflux risk

## Pharyngeal pouch (Zenker's diverticulum)

→ *food accumulates & decomposes*

**SYMPTOMS:** halitosis, regurgitation, bowel sounds in the oesophagus (bubbling/gurgling)

**INVESTIGATIONS:** barium swallow

**MANAGEMENT:** depends on size of pouch

- Stapling of pouch
- Dissection of pouch

## Oesophageal spasm

→ *sudden 'cramping' of muscles*

**SYMPTOMS:** **acute onset pain***

**INVESTIGATIONS:** manometry

**MANAGEMENT:**

- Manage underlying conditions (e.g. GORD)
- Muscle relaxants e.g. sildenafil, Botox, CCBs
- Myotomy (of lower sphincter)

*this pain can mimic cardiac chest pain & often radiates into the jaw

# Oesophageal cancer

→ **UK prevalence:** 14 in 100,000 (increasing due to RFs)
→ **M:F = 2:1**

**Squamous cell (20%):** upper 2/3 oesophagus
RF = **smoking, alcohol,** Asian, achalasia

**Adenocarcinoma (80%):** lower 1/3 oesophagus
RF = **smoking, alcohol,** obesity, Barrett's oesophagus (GORD)

**Presents late & POOR PROGNOSIS**[4]

| Stage | 5y Survival |
|---|---|
| 1 | 53% |
| 2 | 30% |
| 3 | 16% |
| 4 | 0% |
| Overall | 17% |

## Symptoms

- **Progressive dysphagia:** solids → liquids → saliva
- Weight loss & anorexia
- Retrosternal chest pain
- Lymphadenopathy
- ± cough, aspiration, hoarseness

## Investigations

- **OGD & biopsy** – **histological grading**
- **CT chest, abdo, pelvis** – **TNM staging**
- Endoscopic USS – more detailed T&N staging
- PET scan – detects metabolically active mets

**TNM staging**

| Tumour | Nodes | Metastases |
|---|---|---|
| $T_{is}$ – tumour *in situ*<br>$T_{1a}$ – invades lamina propria<br>$T_{1b}$ – invades submucosa<br>$T_2$ – invades muscularis propria<br>$T_3$ – invades adventitia<br>$T_4$ – invades adjacent tissues | $N_0$ none<br>$N_1$ 1–2 LNs<br>$N_2$ 3–6 LNs<br>$N_3$ 7+ LNs | $M_0$ no mets<br>$M_1$ distant mets |

## Management[5]

Management requires an MDT approach

| **Adenocarcinoma** | |
|---|---|
| **$T_{is}$ or $T_{1a}$** ($N_0$, $M_0$) | Endoscopic mucosal resection/dissection (EMR/EMD) |
| **$T_{1b}$** ($N_0$, $M_0$) **or >75yrs** | Surgical resection **or** definitive chemoradiotherapy |
| **$T_2$, $T_3$, $T_4$** ($M_0$) | Neoadjuvant chemo → surgery + adjuvant chemo |
| **Squamous cell carcinoma** | |
| **$T_{1a}$** ($N_0$) | EMR/EMD |
| **All others** ($M_0$) | Neoadjuvant chemo → surgery + adjuvant chemo |
| **Any histology with mets** ($M_1$) = palliative care | |
| 1. Chemo ± radiotherapy<br>2. Symptom relief: stents, analgesia | |

**Complications of upper GI surgery:**

- **Weight loss** – need dietetic support
- **Dysphagia** – due to strictures
- **Reflux**
- **Delayed emptying**

**SURGICAL OPTIONS:**

**Ivor Lewis oesophagectomy** **(2 stage → open or keyhole)**

Stage 1 (abdominal):

- mobilise / free stomach from blood supply

Stage 2 (thoracic):

- mobilise & resect affected part of oesophagus
- pass 'free' stomach through hiatus & staple to remaining oesophagus
- pylorectomy to improve gastric emptying post-op

NB. Oesophageal surgery = high morbidity & mortality
→ need careful pre-op assessment for suitability

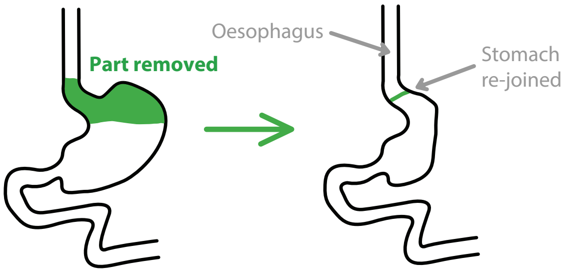

**Fig. 3.2** Ivor Lewis oesophagectomy.

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[4] Cancer survival by stage at diagnosis for England, 2019. ONS.
[5] NICE (2018) *Oesophago-gastric cancer* [NG83]

# Gastric cancer

*prevalence is increasing globally due to increased prevalence of modifiable RFs

**Risk factors:**
→ **Internal:** pernicious anaemia, *H. pylori*, polyps
→ **External:** smoking, high salt/nitrate (red meat)
→ **Genetic:** Japanese, HNPCC, Group A blood

**Presents late & POOR PROGNOSIS**[7]

| Stage | 5y Survival |
|---|---|
| **1** | 65% |
| **2** | 36% |
| **3** | 24% |
| **4** | 0% |
| **Overall** | **20%** |

**Any histology with mets** = **palliative care**

1. Chemo ± radiotherapy
2. Symptom relief: stents, bypass, analgesia

**Gastrectomy complications:**

- Vit B12 / iron deficiency
- Early satiety / weight loss
- Osteoporosis/osteomalacia

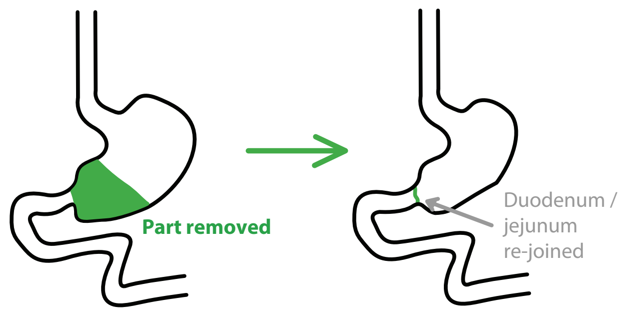

**Fig. 3.3** Distal gastrectomy.

→ **UK prevalence*:** 10 in 100,000 → 5th most common cancer worldwide[6]
→ **M:F = 3:1** → **Peak age: 50–70y**

## Types

1. **Adenocarcinoma (85%)**
2. **Other (15%)** – lymphoma, leiomyosarcoma, GISTs

## Symptoms

- **Epigastric pain / dyspepsia**
- **Early satiety**
- N&V, anorexia, weight loss
- Dysphagia

often non-specific & mimic PUD

## Investigations

- **OGD & multiple ulcer edge biopsies** – **histological grading & location**
- **CT chest, abdo, pelvis** – **TNM staging** (same as oesophageal)
- Endoscopic USS – more detailed T&N staging

## Signs O/E

- **Epigastric mass** (50%)
- Virchow's node enlargement
- Hepatomegaly, ascites
- Jaundice, acanthosis nigricans

only in late stage / metastases

## Management[8]

| Adenocarcinoma | |
|---|---|
| **$T_{is}$ or $T_{1a}$** ($N_0$, $M_0$) | Endoscopic mucosal resection |
| **$T_{1b}$** ($N_0$, $M_0$) **or >75y** | Direct to surgery |
| **$T_2$, $T_3$, $T_4$** ($M_0$) | Neoadjuvant chemo → surgery + adjuvant chemo |

### SURGICAL OPTIONS:

| Siewert class | Location | Management |
|---|---|---|
| **1** | 1–5cm above GOJ | Oesophagectomy |
| **2** | <1cm above or <2cm below GOJ | Oesophagectomy **or** total gastrectomy |
| **3** | 2–5cm below GOJ | Total gastrectomy |
| **Distal** | Near pylorus | Distal gastrectomy |

**Total gastrectomy + Roux-en-Y reconstruction:**

1. Resection of stomach
2. Connect proximal jejunum to lower oesophagus
3. Reconnect distal duodenum further down jejunum to allow passage of bile

**Distal gastrectomy:**

1. Resection of distal part of stomach
2. Various reconstruction options **(Roux-en-Y = better outcome but ↑ risk)**

---

[6] International Agency for Research on Cancer, WHO 2020
[7] Cancer survival by stage at diagnosis for England, 2019. ONS.
[8] NICE (2018) *Oesophago-gastric cancer* [NG83]

# Coeliac disease

Chronic inflammation caused by autoimmune destruction of small intestinal villi in response to gluten

## Clinical presentation

Very common: 1 in 100

- **GI symptoms:** indigestion, bloating, abdo pain, altered bowel habit
- **Rash:** dermatitis herpetiformis
- **Signs of malabsorption:** weight loss, fatigue, anaemia (failure to thrive in children)

**Complications:**
- Osteoporosis
- **Intestinal lymphoma**
- Anaemia
- Neuropathy

## Investigations

- **Hx & examination:** signs of nutrient deficiency (pallor, angular stomatitis etc.)
- **Malnutrition bloods:** anaemia, B12/folate deficiency, bone profile
- **Coeliac antibodies**
  - **IgA tTG (tissue transglutaminase):** >90% specific for coeliac
  - **Serum IgA levels:** some people are IgA deficient so IgA tTG will appear normal
  - **EMA (endomysial antibody):** if tTG only weak positive or if patient IgA deficient
- **Biopsy of D2*:** ↑ intra-epithelial lymphocytes, villous atrophy (flat villi) → **confirms Dx**
  - ↳ (not needed in some centres if tTG levels alone are high enough for diagnosis)

*Patients need to be on gluten-containing diet for 6w before biopsy

## Management[9]

- **Lifelong gluten-free diet**
  - Education & information sources
  - Dietitian input – avoid barley, wheat, rye (can have rice)
- Correct any nutrient deficiencies
- DEXA scan for bone health (repeat every 5y)
- Annual review – diet, weight, tTG, assess for complications (anaemia, nutrient deficiencies)

# Obesity

**13% adults worldwide**
**20–25% adults in UK**

## Complications

| | |
|---|---|
| **Metabolic syndrome** | DM, HTN, hyperlipidaemia |
| **Mechanical** | OA, back pain, incontinence |
| **Malignancy** | Breast, uterus, CRC |
| **Respiratory** | Asthma, sleep apnoea |
| **Abdominal** | Liver cirrhosis, NAFLD, GORD |
| **Psychological** | Low self-esteem |

## Risk factors

- Poor diet
- Hypothyroidism
- Lack of exercise
- Cushing's
- Steroid therapy
- Low self-esteem
- Quitting smoking

| Class | BMI |
|---|---|
| **1 (moderate)** | 30–34.9 |
| **2 (severe)** | 35–40 |
| **3 (v. severe)** | >40 |

**MANAGEMENT: MDT**

1. **Lifestyle:** exercise, diet, CVD risk factors
2. **Medication:** orlistat/semaglutide
3. **Surgery:** ↓ weight first to show engagement
   - gastric band / gastric sleeve
   - diversion procedure e.g. gastro-jejunostomy
   - Roux-en-Y

---

[9] NICE (2020) CKS: *Management of confirmed coeliac disease*

# Acute diarrhoea

**Causes of gastroenteritis:**

**Viral:** norovirus, rotavirus
**Bacterial:** *E. coli, Salmonella, Campylobacter, C. difficile*

## Causes

- Gastroenteritis
- Traveller's diarrhoea – *E. coli, Giardia, Campylobacter, Shigella*
- IBS/IBD
- Coeliac
- Colorectal cancer

**Symptoms by organism**

| | |
|---|---|
| ***E. coli*** | Watery diarrhoea + nausea + cramps |
| ***Giardia*** | Upper GI Sx (bloating/belching) |
| ***Campylobacter*** | Bloody diarrhoea + cramps + vomiting |
| ***Salmonella*** | |

## Investigations

Aim = to r/o other causes of symptoms

1. **Hx & exam:** recent travel, diet change, infectious contacts, immunosuppressants, weight loss, recent antibiotics
2. **Bloods**
   - FBC – anaemia in malignancy, IBD, coeliac
   - ESR/CRP – infection/IBD
   - U&Es – show **dehydration**
   - LFTs, glucose, clotting
   - Anti-tTG/EMA antibodies: r/o coeliac
3. **Stool sample:** MCS, faecal calprotectin*
   (+ ova, cysts, and parasites if travel history indicates)

*NB. faecal calprotectin also raised in bacterial/viral infection

## Management

1. **Assess for dehydration:** oral rehydration (IV if sustained D&V)
2. **Loperamide** – if short-term relief needed
3. **ABX** – not recommended unless severe systemic upset
4. **Public health** – notifiable disease/time off work if food industry

**_C. difficile_ = Gram +ve bacillus**

→ **common hospital-acquired infection (usually 5–10d after ABX)**

**Symptoms: 5% = asymptomatic carriers**
- Watery diarrhoea + systemic upset
- May progress to fulminant colitis & toxic megacolon

**Investigation:**
- **stool sample**

**Management:**
**Mild:** PO metronidazole
**Severe:** PO vancomycin
(IV if non-responsive)

**Risk factors:**
- **prolonged/multiple ABX use or PPI**
- **prolonged hospital stay**
- increasing age
- immunosuppression
- NG tube / invasive gastro procedure

3rd line Tx: faecal microbiota transplant

# Irritable bowel syndrome

Up to 20% of population
F:M = 2:1
Peak onset 20–30y

= group of **abdominal symptoms** for which **no organic cause** is found

## Diagnostic criteria

**≥6m of abdominal pain/discomfort**
→ relieved on defecation **OR**
→ associates with altered bowel frequency / stool form
**and**
**≥2 of the following:**

- bloating/distension
- passage of mucus
- incomplete evacuation / straining / urgency
- symptoms worsened by eating

± **non-intestinal symptoms** e.g. urinary, headache, fatigue, back pain, dysmenorrhoea

## Investigations

- Careful history and physical examination
- FBC, ESR/CRP – r/o IBD
- Faecal calprotectin – r/o IBD
- Coeliac screen – endomysial antibodies (EMA) / tissue transglutaminase (tTG) antibodies
- CA 125 – for older women to r/o ovarian cancer

## Risk factors / causes

- Stress, anxiety, depression – including childhood trauma/abuse
- Gastroenteritis – precedes up to 30% of cases
- Antibiotics
- Eating disorders
- Trauma/surgery

## Management[10]

### 1. DIET/LIFESTYLE

- **Low FODMAP diet** – limit insoluble fibres & sugars that trigger bloating → ideally should be dietitian-guided
- **Regular meals,** without long gaps between
- At least 8 cups of **fluid per day but avoid caffeine** / fizzy drinks
- **Regular exercise**

### 2. PSYCHOLOGICAL

- CBT, hypnotherapy, acupuncture, herbal therapies

### 3. PHARMACOLOGICAL

- **Constipation:** laxatives (avoid lactulose as worsens bloating)
- **Diarrhoea:** antidiarrhoeals (loperamide)
- **Abdominal pain/cramps:** antispasmodics (mebeverine/hyoscine butylbromide)
- **Chronic/resistant pain:** low dose TCAs (SSRIs, gabapentin, pregabalin = 2nd line)

**Main types of symptom:**

**A**bdo pain/discomfort
**B**loating
**C**hanged bowel habits

**MUST SCREEN FOR CANCER RED FLAGS:**

- Weight loss
- Melaena
- Altered bowel habit >60y
- FHx colon cancer <50y
- Abdo or rectal mass

**Need 2ww referral for colonoscopy**

**Prognosis**

- Not associated with any serious long-term disease
- Symptoms may fluctuate in severity

**FODMAP diet**

**F**ermentable – wheat/rye
**O**ligosaccharides – legumes
**D**isaccharides – fruit & veg
**M**onosaccharides – milk, yoghurt
**A**nd – soft cheese
**P**olyols – sweeteners

**Follow-up**

- Agreed between clinician & patient dependent on symptoms/response to treatment etc.
- **Safety-net for red flags of bowel cancer**

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[10] NICE (2008, updated 2017) *Irritable bowel syndrome in adults: diagnosis and management* [CG61]

# Inflammatory bowel disease

Due to **dysregulated immune inflammatory response → genetic predisposition + environment**

<table>
<tr><th></th><th>Crohn's<br>M = F</th><th>Ulcerative colitis<br>F > M</th></tr>
<tr><td rowspan="2">**Epidemiology**</td><td colspan="2">Peak age 25–30y</td></tr>
<tr><td>Smoking **increases** risk</td><td>Smoking **decreases** risk</td></tr>
<tr><td>**Location**</td><td>Mouth → anus</td><td>Colon/rectum only</td></tr>
<tr><td>**Anal involvement**</td><td>Often (fistula, haemorrhoids)</td><td>Never</td></tr>
<tr><td>**Continuity**</td><td>**Discontinuous** 'skip lesions'</td><td>**Continuous** proximal spread from rectum</td></tr>
<tr><td>**Histology**</td><td>• **Transmural**<br>• Deep ulcers (cobblestone appearance)<br>• Granulomas<br>• Goblet cells present</td><td>• **Mucosa only**<br>• Superficial ulcers<br>• Pseudopolyps<br>• Crypt abscesses (filled with neutrophils)<br>• Few goblet cells present</td></tr>
<tr><td>**Symptoms**</td><td colspan="2">→ **Abdo pain** (may be crampy)<br>→ **Weight loss** (anaemia & B12 deficiency)<br>→ **Diarrhoea** (can be bloody)<br>→ **Tenesmus** (in ulcerative colitis)</td></tr>
<tr><td rowspan="2">**Extra-GI signs**</td><td colspan="2">• Arthritis, uveitis/iritis<br>• Erythema nodosum<br>• Pyoderma gangrenosum<br>• Venous thrombosis<br>• Fatty liver<br>• Osteoporosis<br>• **Ankylosing spondylitis***<br>*Autoimmune conditions are not improved by controlling disease activity with medication/surgery</td></tr>
<tr><td>• Mouth ulcers<br>• Anal fistulae, tags, fissures<br>• Sometimes PSC (but much less than with UC)</td><td>• **Primary sclerosing cholangitis (PSC)***<br>• **Cholangiocarcinoma***</td></tr>
<tr><td>**Investigations**</td><td colspan="2">1. **Bloods**<br>• FBC, iron studies = anaemia & B12 deficiency<br>• CRP/ESR = raised<br>2. **Stool samples**<br>• MCS × 3 – r/o infection<br>• Faecal calprotectin = sensitive for colonic inflammation<br>• Stool chart = **specific for IBD**<br>3. **Radiology**<br>• AXR = lead-piping & shows complications **such as toxic megacolon**<br>• CT abdomen<br>4. **Sigmoid/colonoscopy or endoscopy + biopsy**<br>*May cause bleeding as it disrupts inflamed mucosa (esp. in UC)*</td></tr>
<tr><td>**General education**</td><td colspan="2">1. Information on condition – relapsing & remitting → lifelong<br>2. Nutrition & diet<br>3. Smoking cessation<br>4. Medication adherence & side-effects<br>5. Disease complications</td></tr>
</table>

| | Crohn's | Ulcerative colitis |
|---|---|---|
| **Follow-up & monitoring** | → Colonoscopic surveillance for cancer (begins 10y after diagnosis)<br>→ Anaemia, osteopenia & fracture risk | |
| **Management of acute flares**[11] | **MILD:**<br>Oral prednisolone/budesonide<br>→ Tapered discontinuation of steroids & R/V<br>**MODERATE/SEVERE: ADMISSION**<br>**1st line:** IV hydrocortisone<br>**2nd line:** azathioprine<br>(if contraindicated, consider methotrexate)<br>**3rd line:** infliximab (or other biologics)<br>→ Transfer to oral prednisolone ASAP | **MILD:**<br>5-ASA (mesalazine) PO ± PR daily<br>**MODERATE / resistant to Mild Tx:**<br>1. **Induce remission:** PO prednisolone + 5-ASA<br>2. **Maintain remission:** 5-ASA (mesalazine)<br>**SEVERE / ≥2 courses of steroids per year: ADMISSION**<br>1. **Induce remission:**<br>IV hydrocortisone<br>**or** calcineurin inhibitors (ciclosporin)<br>2. **Maintain remission:**<br>Immunosuppressants (azathioprine)<br>**or** biologics (infliximab) |
| **Long-term management** | **1st line:** immunosuppressant (e.g. azathioprine)<br>**2nd line:** biologics (or methotrexate)<br>→ Oral metronidazole for peri-anal disease | **1st line:** 5-ASA (mesalazine)<br>**2nd line:** oral thiopurines (e.g. azathioprine) |
| **Role of surgery** | **Never curative (still need maintenance Tx)**<br>→ Temporary ileostomies – rest distal bowel<br>→ Limited resection of severely affected parts | **Can be curative**<br>→ Colectomy |

## Assessing severity of IBD

Severity assessment guides Tx.

**Truelove & Witts' criteria**

| | Mild | Moderate | Severe* |
|---|---|---|---|
| **Bowel movements per day** | <4 | 4–6 | ≥6 |
| **Blood in stools** | None/small amounts | Moderate–severe amounts | Clearly visible |
| **Pyrexia (>37.8°C)** | No | No | Yes |
| **Heart rate >90bpm** | No | No | Yes |
| **Anaemia** | No | No | Yes |
| **ESR** | ≤30 | ≤30 | >30 |

***Severe disease** if ≥6 bowel movements per day PLUS ≥1 feature of systemic upset

## Options for surgery

1. **Panproctocolectomy ± ileo-anal pouch**
   → **PROs:** curative in UC & prevents malignant change in rectum
   → **CONs:** permanent stoma & extensive pelvic dissection
2. **Subtotal colectomy:** leaves rectal stump
   → **PROs:** quicker/lower risk
   → **CONs:** rectal stump needs monitoring for malignancy

**Indications for surgery:**
- Failed medical therapy / recurrent disease
- Severe disease / very unwell
- Complications (adhesions, strictures, bowel obstruction)

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[11] NICE Clinical Scenario (2020) *Confirmed Crohn's disease*; (2020) *Confirmed ulcerative colitis*

## Complications of inflammatory bowel disease

<table>
<tr><th></th><th>Crohn's</th><th>Ulcerative colitis</th></tr>
<tr><td rowspan="2"><b>Surgical complications of IBD</b></td><td colspan="2">• Perforation & abscess formation<br>• Chronic GI bleed <b>or</b> acute GI bleed</td></tr>
<tr><td>• Adhesions & strictures<br>• Peri-anal disease / fistulae (in 50%)<br>• Bowel obstruction<br>• Colorectal cancer</td><td>• Toxic megacolon<br>• Colorectal cancer</td></tr>
</table>

**Management of generalised perforation** (peritonitis)

1. **ABCDE** – fluid resuscitation, catheter, G&S
2. **IV ABX**
3. **Escalate & assess fitness for EMERGENCY SURGERY**

**Management of localised perforation** (abscess)

1. **ABCDE** – fluid resuscitation, catheter, G&S
2. **USS/CT guided drainage + IV ABX**
3. **Optimise for surgery** (e.g. NG tube for nutrition)
4. **Imaging & planning of surgery**

## Managing bowel obstruction in IBD (due to strictures)

→ may present more gradually with weight loss / anorexia

### TYPES OF STRICTURE:

1. **Inflammatory:** oedema of wall causes narrowed lumen
2. **Fibrotic:** repeated inflammation has resulted in scarring

### MANAGEMENT:

1. **Inflammatory stricture:** trial medical therapy → IV hydrocortisone (settle inflammation) → biologics
2. **Fibrotic stricture:**
   - **1st line:** endoscopic decompression & stenting → balloon dilatation
   - **2nd line:** surgery (strictureplasty or resection + anastomosis / stoma formation)

*Strictureplasty = preferred as preserves bowel length*
*BUT resection indicated if multiple strictures*

## Toxic megacolon

→ severe inflammation causes wall thinning & huge dilation (>6cm) with toxin build-up

**Clinical presentation:** **severely unwell**

- Fever, tachycardia, shock
- Distension & abdo pain

**Management:** **urgent surgery**

- Risk of perforation

## Colorectal cancer in IBD

- Accounts for 10% of IBD mortality
- **5–10% risk** after 20y
- Inflammatory lesions = **pre-neoplastic**

**Surveillance: colonoscopy → start 10y after Dx**
Low risk = 5-yearly
Moderate risk = 3-yearly
High risk = annually

**Risk factors for colorectal cancer in IBD:**

- Longer disease duration
- Extent & severity of disease
- Presence of pseudopolyps
- Primary sclerosing cholangitis
- Family Hx

# HEPATO-PANCREATO-BILIARY

# 04

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## ABBREVIATIONS

**AFP** – Alpha fetoprotein
**ALP** – Alkaline phosphatase
**ALT** – Alanine aminotransferase
**APCKD** – Adult polycystic kidney disease
**AST** – Aspartate aminotransferase
**BR** – Bilirubin
**CBD** – Common bile duct
**CBR** – Conjugated bilirubin
**CCK** – Cholecystokinin
**CEA** – Carcinoembryonic antigen
**CLD** – Chronic liver disease
**CMV** – Cytomegalovirus
**Cu** – Copper
**DEXA** – Dual energy X-ray absorptiometry
**DIC** – Disseminated intravascular coagulation
**EBV** – Epstein–Barr virus
**ERCP** – Endoscopic retrograde cholangiopancreatography
**Fe** – Iron
**GB** – Gallbladder
**GGT** – Gamma-glutamyl transferase
**GORD** – Gastro-oesophageal reflux disease
**HBV** – Hepatitis B virus
**HPV** – Hepatic portal vein
**IBD** – Inflammatory bowel disease
**INR** – International normalised ratio
**MRCP** – Magnetic resonance cholangiopancreatography
**MTX** – Methotrexate
**NAFLD** – Non-alcoholic fatty liver disease
**NBM** – Nil by mouth
**OCP** – Oral contraceptive pill
**OGD** – Oesophago-gastroduodenoscopy
**OTC** – Over the counter
**PBC** – Primary biliary cirrhosis
**PSC** – Primary sclerosing cholangitis
**PT** – Prothrombin time
**PUD** – Peptic ulcer disease
**RA** – Rheumatoid arthritis
**RIF** – Right iliac fossa
**RUQ** – Right upper quadrant
**SBP** – Spontaneous bacterial peritonitis
**SCA** – Sickle cell anaemia
**SLE** – Systemic lupus erythematosus
**STI** – Sexually transmitted infection
**TB** – Tuberculosis
**TIPS** – Transjugular intrahepatic portosystemic shunt
**UCBR** – Unconjugated bilirubin

# Hepatitis

**Criteria of acute liver failure:**

1. **Increase in PT** by 4–6sec (INR >1.5) **and**
2. Development of **hepatic encephalopathy (HE)**
3. **Without pre-existing cirrhosis** & illness <6m

**Paracetamol overdose:**

**Sx:** RUQ pain, confusion, N&V
**Ix:** ↑ ALT (>1000)
**Mx:** ABCDE, *N*-acetylcysteine ± liver transplant (if severe)

**Acute alcohol binge (alcoholic hepatitis):**

**Sx:** RUQ pain, jaundice, N&V, systemic upset ± signs CLD
**Ix:** ± isolated ↑ GGT (alcoholic liver disease)
**Mx:** ± prednisolone

**Acute viral hepatitis:**

**Sx:**
- Pre-icteric: fever, malaise, anorexia, nausea
- Icteric: RUQ pain, jaundice

**Ix:** ↑ ALT (>1000), Igs (for hepatitis, EBV etc.)
**Mx:** supportive + antivirals (seek senior advice)

**Autoimmune hepatitis:**

**RF:** young women / peri-menopausal, other autoimmune conditions
**Sx:** non-specific (fatigue, arthralgia, weight loss)
**Ix:** ↑ ALT (>1000), autoantibodies (ANA, anti-mito, anti-SM)
**Dx:** liver biopsy
**Mx:** immunosuppression (steroids + azathioprine) ± transplant

Hepatitis = inflammation of the liver
Chronic liver disease = inflammation >6m

## Pathology

**Acute:** liver insult → inflammatory immune response → cytotoxic cell destruction → necrosis
**Chronic:** if **inflammation** continues >6m = **fibrosis → nodular regeneration → cirrhosis = chronic liver disease**
**Stage:** degree of inflammation
**Grade:** degree of fibrosis & cirrhosis

## Causes

### 1. ACUTE HEPATITIS

*Hepatitis B & C can cause chronic disease

| | |
|---|---|
| **Infection** | • Hepatitis A, B*, C*, D, E<br>• EBV, CMV, TB |
| **Drugs**<br>Most<br>Normal<br>People<br>Cannot<br>Function<br>If<br>On<br>Cocaine | <br>Methyldopa /MTX<br>Nitrofurantoin<br>Paracetamol<br>Chlorpromazine<br>Flucloxacillin<br>Isoniazid<br>OCP<br>Co-amoxiclav |
| **Autoimmune** | • Hepatitis<br>• Primary biliary cholangitis<br>• Primary sclerosing cholangitis |
| **Metabolic** | • Wilson's (↑ Cu)<br>• Haemochromatosis (↑ Fe)<br>• Alpha-1-antitrypsin deficiency |
| **Alcohol** | • Binge drinking |

### 2. CHRONIC HEPATITIS / CHRONIC LIVER DISEASE

Most common causes

| | |
|---|---|
| **Alcohol** | Chronic excess |
| **NAFLD** | Obesity, DM, ↑ cholesterol |
| **Infection** | Hep B, C |
| **Chronic drug use** | • Methyldopa<br>• Methotrexate<br>• Nitrofurantoin<br>• Chlorpromazine |
| **Autoimmune** | • Hepatitis<br>• Primary biliary cholangitis<br>• Primary sclerosing cholangitis |
| **Metabolic** | • Wilson's (↑ Cu)<br>• Haemochromatosis (↑ Fe)<br>• Alpha-1-antitrypsin deficiency |

## General presentation

### ACUTE HEPATITIS:

- Anorexia, nausea, vomiting
- RUQ pain
- ± systemic upset

**ALT >1000 in:**

1. Hypotension
2. Drug insult
3. Viral hepatitis
4. Autoimmune

### CHRONIC HEPATITIS:

Features of cirrhosis:
**Non-specific:** anorexia, weight loss, fatigue, clubbing
**Abnormal clotting:** bruising / ↑ INR
**Hyperaldosteronism (↓ hormone breakdown):** feminisation, ↓ $K^+$, ↑ BP
**Portal hypertension:** hepatosplenomegaly, spider naevi, ascites
**Encephalopathy:** drowsy/confused

Features of decompensated cirrhosis: **jaundice, ascites, encephalopathy** (after alcohol binge, hepatotoxic drugs, infection, SBP, variceal bleed)

## Investigations

### 1. HISTORY & ABDO EXAM:

- PMHx, DHx, FHx
- Autoimmune conditions
- RF for viral hepatitis: blood transfusion, IVDU, tattoos, unprotected sex, travel
- Alcohol & diet / T2DM

### 2. BLOOD TESTS

| Acute | Liver screen |
|---|---|
| • Toxin screen (drugs/alcohol) | • FBC – infective markers<br>• LFTs<br>• U&Es<br>• Viral serology<br>• Ceruloplasmin (Wilson's)<br>• Transferrin/ferritin sats<br>• Autoantibodies (ANA, anti-SM, IgG) |
| **Chronic/cirrhosis** | |
| • Albumin, BR, clotting (PT) – show synthetic function<br>• Fe/Cu – metabolic condition<br>• AFP – hepatic carcinoma marker<br>• USS/Fibroscan (measures fibrosis & fatty change)<br>• Liver biopsy* | |

***Liver biopsy** = **Gold standard** for staging & grading **but invasive**

| Indications: | Contraindications: |
|---|---|
| • Confirm Dx & prognosis<br>• Suspected neoplasm<br>• Unexplained jaundice | • Clotting disorder<br>• Profound anaemia<br>• Marked ascites<br>• Subphrenic infection |

## Management of chronic liver disease / cirrhosis[1]

1. **Address underlying cause:** e.g. ↓ alcohol consumption / fatty diet, tight diabetic control
2. **Assess for complications:** USS & AFP for carcinoma, upper endoscopy for varices
3. **Manage complications:** anaemia, disordered clotting, varices, ascites, encephalopathy, SBP (spontaneous bacterial peritonitis)
4. **Regular R/V:** surveillance (liver USS ± AFP) for decompensation/malignancy
5. **Calculate MELD score** (model of end-stage liver disease) biannually

AST:ALT ratio in alcoholic hepatitis = 2:1

**Liver function tests:**

**ALT/AST:** ↑ in parenchymal disease (10–40U/L)
**ALP/GGT:** ↑ in bile duct obstructions (<130U/L)
**Albumin:** ↓ in CLD (3.5–5g/dL)

**Bilirubin:** ↑ in CLD
- **Direct (conj.):** intra/post-hepatic jaundice
- **Indirect (unconj.):** pre-hepatic jaundice

**Causes of ↑ ALP**

**Placenta, Liver, Kidney, Bones**
('**Pl**ate of **Liver** & **Kidney B**eans')

**Interpretation of viral serology:**

| | Acute HBV | Chronic HBV | Cleared HBV | Vaccinated |
|---|---|---|---|---|
| **HBcAb IgM** | + | – | – | – |
| **HBcAb IgG** | + | + | + | – |
| **HBsAg** | + | + | – | – |
| **Anti-HBs** | – | – | + | + |
| **HBeAg** | + | +/– | – | – |
| **Anti-HBe** | – | +/– | +/– | – |
| **HBV DNA** | High | Low | – | – |

**Ascites** managed with diuretics, drainage & TIPS (transjugular intrahepatic portosystemic shunt)

Prophylactic antibiotics (rifaximin) for **encephalopathy** or **upper GI bleeding**

**PORTAL HYPERTENSION** → ↑ **pressure in hepatic portal vein** causes **backflow** through portosystemic anastomoses

**Causes:**

| Pre-hepatic | Hepatic | Post-hepatic |
|---|---|---|
| • Splenic vein / HPV thrombosis<br>• Congenital atresia/stenosis<br>• Extrinsic compression | • **Cirrhosis (90%)**<br>• Schistosomiasis<br>• Drugs | • Right ventricle failure<br>• Budd–Chiari syndrome |

**Complications:**

1. **Varices (oesophageal, rectal, para-umbilical)**
   → *Prophylaxis:* beta-blockers (carvedilol) → banding if BBs contraindicated
   → *Mx of bleed:* ABCDE, IV ABX, terlipressin → OGD for Dx & banding
2. **Ascites**
3. **Hepatosplenomegaly**
4. **Encephalopathy** – toxins bypass liver

**Blood supply of liver:**
**75% HPV**
**25% Hepatic artery**

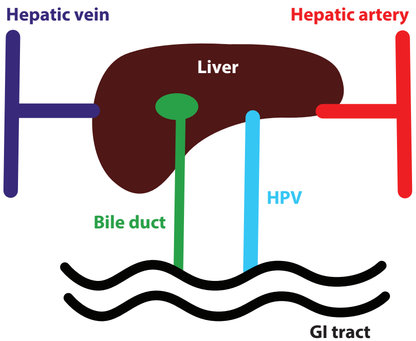

**Fig. 4.1** Liver blood supply.

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[1] NICE (2016) *Cirrhosis in over 16s* [NG50]

# Ascites

**Risk factors for ascites:**
- Known liver disease
- FHx of liver disease
- Alcohol
- Hepatotoxic drugs
- Obesity, T2DM, ↑ lipids
- Autoimmune disease
- Heart failure
- GI malignancies
- RFs for viral hepatitis (IVDU, tattoos, STIs)

**Emergency presentation:**
- Abdominal tenderness (peritonitis/SBP)
- Abdominal mass (malignancy)
- Fever/sepsis (SBP)
- Drop in haematocrit (haemoperitoneum)

**Assessing ascitic fluid:**
- Cell count & differential
- Albumin*
- Total protein

**Measure serum albumin simultaneously*

*Must measure platelet count & prothrombin time before paracentesis

An abnormal collection of fluid in the peritoneal cavity

## Causes

| Conditions causing portal hypertension | Unrelated to portal hypertension |
|---|---|
| • **Liver cirrhosis:** 75% of cases<br>• **Congestive heart failure**<br>• **Malignant:** liver mets, ovarian cancer<br>• Constrictive pericarditis<br>• Alcoholic liver disease<br>• Budd–Chiari syndrome | • **Infection:** TB, HIV, chlamydia, fungal<br>• **Malignant:** peritoneal carcinoma<br>• **Hypoalbuminaemia:** nephrotic syndrome<br>• **Other:** SLE |

## Signs/symptoms

- Abdominal distension
- Dyspnoea & early satiety
- Shifting dullness
- Stigmata of cirrhosis (palmar erythema, spider naevi, jaundice, hepatosplenomegaly, ↑ JVP)

## Investigations

- USS abdomen – confirms diagnosis
- Doppler USS – assess patency/thrombosis of portal veins
- Routine bloods – LFTs, U&Es, FBC, metabolic screen, clotting screen
- Hepatitis serology
- **Abdominal paracentesis*** – for **new onset ascites or suspicion of SBP**
- CT/MRI – particularly if suspect malignant cause
- Liver biopsy – assess stage and grade of cirrhosis

## Management[2]

***Mx is based on severity** (graded 0–4 depending on serum ferritin levels)*

1. **Treat emergency presentations:** hepatorenal syndrome, SBP etc. (see below)
2. **Diuretics:** spironolactone monotherapy (+ furosemide if refractory ascites)
3. **Albumin solution infusion:** to be considered after therapeutic paracentesis
4. **Salt- & fluid-restricted diet:** 1–1.5L per day if hypervolaemic hyponatraemia
5. **TIPS (transjugular intrahepatic portosystemic shunt):** consider in refractory ascites
6. **Liver transplant:** consider in refractory ascites / severe cirrhosis / hepatorenal syndrome

**Complications of ascites:**
- **Spontaneous bacterial peritonitis (SBP)** = infection of ascitic fluid
  **Sx:** fever & extreme abdominal tenderness
  **Ix:** ascitic tap: neutrophils >250, cultures to guide ABX therapy
  **Mx:** IV ABX
- **Hepatorenal syndrome** = rapid deterioration in renal function secondary to liver cirrhosis
  **Mx:** IV albumin + terlipressin → consider for urgent liver transplant
- **Hepatic encephalopathy** = altered sleep, increased confusion, coma
  **Mx:** urgent Tx with lactulose & rifaximin
- **Refractory ascites** = not controlled with maximum dose diuretics
  **Mx:** large volume paracentesis + albumin replacement → consider TIPS or liver transplant

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[2] British Society of Gastroenterology (2021) *Guidelines on the management of ascites in cirrhosis*

# Liver neoplasms

↳ Malignant lesions are rare

→ Majority of liver lesions are benign

## Types of liver neoplasm

| Benign (F>M) | Malignant (M>F) |
|---|---|
| 1. Haemangioma (5%)<br>2. Focal nodular hyperplasia (FNH) (3%)<br>3. Adenoma (oestrogen-driven)<br>4. APCKD<br>DDx: cysts/abscess | **90% = 2° metastases**<br>lung, stomach, colon, breast, uterus<br>**10% = 1° carcinoma**<br>1. Hepatocellular (90%)<br>2. Cholangiocarcinoma |

## Symptoms

- RUQ pain
- Ascites/jaundice (late signs / duct compression)
- **Systemic:** fever, malaise, weight loss, anorexia

benign tumours
= rarely symptomatic

## Investigations

- **Bloods** – FBC, CRP, LFT, U&Es, clotting
- **Tumour markers** – AFP (hepatocellular), CA19-9 (cholangiocarcinoma and pancreatic cancer)
- **Synthetic function:** bilirubin, albumin, PT
- **Radiology:**
  - USS/CT (shows if solid or cystic)
  - **MRI** (shows if benign, malignant or metastatic)
- **Biopsy** – only if radiology is inconclusive

MRI = gold standard

**Risk factors for carcinoma:**
→ **Cirrhosis** (NAFLD, chronic hepatitis)
→ *Aspergillus*
→ Metabolic liver condition
→ Anabolic steroids
→ Parasites

## Management

**Benign:** if asymptomatic, often no Tx needed → FNH & adenomas can bleed / cause pain & need Tx

**Malignant:** depends on TNM stage, liver function, patient status
→ radiofrequency ablation, surgery & chemotherapy

### Liver abscesses

**Symptoms:** anorexia, fever, malaise, RUQ pain

**Causes:** gallstones, diverticulitis, appendicitis, amoebic infection (sheep farming)

**RFs:** DM, immunocompromised, elderly

**Ix:** USS, CT, MRI

**Mx:**
1. 6w ABX
2. Aspiration & drainage
3. ± surgery to treat cause

**Organisms:**
- *Strep. milleri*
- *Strep faecalis*
- *E. coli*
- *Klebsiella*
- Proteus
- Entamoeba

**Liver surgery:** **hepatectomy**
1. Control inflow of blood to segment
2. Control outflow of blood to segment
3. Resect segment/s

→ Must leave at least 2 segments

### Simple liver cysts

**Common**
→ **Most** = asymptomatic & no Tx needed
→ **Large** = pain, mass, anorexia, haemorrhage
(laparoscopic deroofing / open excision + drainage)

# Chronic pancreatitis

→ Repeated bouts of acute pancreatitis
→ Fibrosis & calcification

## Symptoms

1. **Weight loss** (malabsorption)
2. **Steatorrhoea** (↓ lipolysis)
3. **Diabetes mellitus** (↓ insulin secretion)
4. **Upper abdo pain** (in some)

## Investigation

- **Bloods:** FBC, U&Es, clotting, LFT (↑ conj. BR), faecal elastase (exocrine func.)
- **Tumour markers:** CA19-9
- **Abdo USS:** pancreatic mass / dilated common bile duct, ascites
- **Endoscopic USS & biopsy** (+ staging & grading)
- **Contrast-enhanced CT:** confirm Dx and staging

USS & biopsy = DIAGNOSTIC

**Risk factors for chronic pancreatitis:**
→ **Alcohol** (80%)
→ Duct obstruction – strictures, tumours, CF*
→ Hereditary
→ Idiopathic (20–30%)

*If no obvious cause consider CF

**DDx: PANCREATIC NEOPLASM**
Need Ix to exclude this: CA19-9 may be helpful but –ve markers DO NOT r/o pancreatic cancer

# Haemochromatosis

Autosomal recessive disorder of increased iron absorption and release

## Symptoms

- **General:** fatigue, weakness, arthralgia, ↓ libido
- **Skin:** bronzed/pigmented appearance

## Investigations

- Serum transferrin saturation = raised (>45%)
- Ferritin – raised (note it is also raised in acute inflammation)
- Fasting glucose – high levels (indicate pancreatic damage)
- Testosterone, FSH, LH – low levels
- Liver MRI / biopsy / LFTs – assess liver damage / cirrhosis
- ECG & echo – assess cardiac damage
- DEXA scan – if concurrent risk factors for osteoporosis
- Genetic screening – can confirm diagnosis

## Management[3]

*Mx is based on severity (graded 0–4 depending on serum ferritin levels)*

1. **Lifestyle:** avoid iron & vitamin C supplements, ↓ alcohol, hepatitis vaccines
2. **Phlebotomy:** if stage 2 or above (start weekly then transition to less frequent for maintenance)
3. **Iron chelation therapy:** if phlebotomy is contraindicated (e.g. anaemia, severe heart disease)
4. **Monitoring:**
   → Serum ferritin & transferrin saturation 1–3 yearly if stage 0 or 1, more often if 2 or above
   → Yearly hearing and vision checks
   → Liver USS and AFP every 6m if known cirrhosis

**Complications:** if excess iron deposition in organs
- **Cardiac:** cardiomyopathy, arrhythmias
- **Sex organs:** hypogonadism, impotence
- **Pancreas:** diabetes mellitus
- **Liver:** hepatomegaly, carcinoma
- **Bone:** reduced bone density / osteoporosis

**Risk factors for haemochromatosis:**
- Male
- Middle-aged
- White
- FHx
- Iron supplements

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[3] BMJ Best Practice (2022) *Haemochromatosis*

# The spleen

**Functions** = RBC breakdown, lymphocyte synthesis & reservoir

## Causes of splenomegaly

| | |
|---|---|
| **Vascular** | Portal hypertension |
| **Infection** | Infective endocarditis, sepsis, EBV, TB, malaria, schistosomiasis |
| **Inflammation** | RA, SLE, sarcoidosis |
| **Haematological** | Haemolytic anaemia, leukaemia, lymphoma, myeloproliferative disease |
| **Other** | Malignancy, Gaucher's disease (storage disorder) |

## Symptoms of splenomegaly

**Symptoms of PANCYTOPENIA*:**

- Anaemia (pallor/SOB)
- Infection
- Bleeding/bruising

*Splenomegaly causes *hypersplenism* → increased sequestration & destruction of blood cells → PANCYTOPENIA

## Investigations for splenomegaly

*→ Ix aim to determine cause (which can then be treated)*

1. **Hx & exam:** dull to percuss (into RIF), cannot get above (unlike renal mass)
2. **Bloods:** FBC, ESR/CRP, LFT, U&Es → LN or bone marrow biopsy
3. **Imaging:** abdo USS/CT

## Indications for splenectomy

- Trauma (± rupture)
- Spontaneous rupture (EBV)
- Hypersplenism **causing pancytopenia**
- Malignancy (leukaemia/lymphoma)
- Splenic cysts

## Post-splenectomy care

1. **Initial risk of VTE:** LMWH, aspirin & early mobilisation
2. **Lifelong susceptibility to infection*:** immunisations, lifelong penicillin V, carry alert card/bracelet (especially capsulated organisms)

pneumococcal, MenC, HiB,
annual flu vaccine

**Cause of splenic rupture:**

**Blunt trauma** (immediate or delayed)
↑ risk if underlying splenomegaly

↓

*Presents as massive acute bleed **or** peritonism from progressive bleed & eventual shock*

*Warn of risk & careful prophylaxis for malaria if travelling

# Jaundice

**Hyperbilirubinaemia >20μmol/L or >1.2mg/dL**; signs include **yellow skin, sclera and mucosa**; **pruritus** (especially at night) & **fatigue**

### Enterohepatic circulation of bilirubin

1. Haemolysis in spleen forms UCBR
2. UCBR bound to albumin in blood (transported to liver)
3. **Conjugated in liver by gluconyl transferase**
4. CBR excreted via bile into small intestine
   → stercobilin in faeces **or**
   → enterohepatic recycling
5. Recycled urobilinogen transported to kidneys & excreted in urine as urobilin

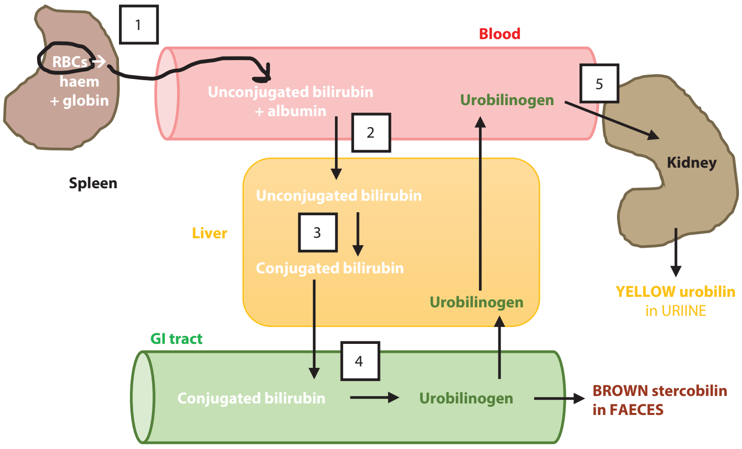
1
RBCs →
haem
+ globin
Spleen
Blood
Unconjugated bilirubin
+ albumin
2
Urobilinogen
5
Kidney
Liver
Unconjugated bilirubin
3
Conjugated bilirubin
Urobilinogen
YELLOW urobilin
in URIINE
GI tract
4
Conjugated bilirubin
Urobilinogen
BROWN stercobilin
in FAECES

**Fig. 4.2** Enterohepatic circulation of bilirubin.

## Causes of jaundice

| **Pre-hepatic**<br>= unconjugated (normal stools/urine) | **Intra-hepatic**<br>= conjugated (pale stools/dark urine) | **Post-hepatic**<br>= conjugated (pale stools/dark urine) |
|---|---|---|
| 1. **↑ Haemolysis:**<br>• Malaria, DIC<br>• Haemolytic anaemias, spherocytosis<br>2. **↓ Liver uptake:** drugs (rifampicin)<br>3. **Eponymous syndromes:**<br>• Gilbert's (no gluconyl transferase)<br>• Crigler–Najjar syndrome | **I**nfection (viral hepatitis, CMV, EBV)<br>**A**utoimmune hepatitis<br>**M**etastases/abscess<br>**D**ecompensated cirrhosis<br>**A**lcohol<br>**D**rugs (paracetamol, statin, isoniazid)<br>**'I AM DAD'** | 1. **Gallstones** (in common bile duct)<br>2. **Pancreatic cancer** (painless)<br>3. **PBC/PSC**<br>4. **Drugs:** steroids, sulphonylureas<br>5. **External bile duct compression**<br>• Lymph nodes/cysts e.g. lymphoma<br>• Cholangiocarcinoma<br>• Mirizzi syndrome |

### Interpreting LFTs

| | Pre-hepatic | Intra-hepatic | Post-hepatic |
|---|---|---|---|
| **Bilirubin** | Normal / ↑ | ↑ | ↑↑ |
| **ALT/AST** | Normal | ↑↑↑ | ↑ |
| **ALP** | Normal | ↑ | ↑↑↑ |

## Investigating jaundice

### 1. HISTORY:

- IVDU/piercings/tattoos
- Blood transfusions before 1980
- Travel, sexual activity, alcohol
- Medications (OTC, steroids etc.)
- FHx / autoimmune conditions

### 2. EXAMINATION:

- Signs of jaundice
- Signs of infection (fever, LNs etc.)
- Signs of chronic liver disease / cirrhosis
- Signs of gallbladder disease (pain, N&V)
- Hepatosplenomegaly / palpable gallbladder

### 3. BLOODS:

- FBC, blood films, clotting
  ↑ reticulocytes = haemolysis
- CRP/ESR – infection/ inflammation
- LFTs – incl. total, unconj. & conj. BR
- U&Es – urinary urobilinogen
- Glucose ↑ in pancreatic damage, ↓ in liver failure
- Hepatitis serology
- Autoantibodies / alpha-1 antitrypsin
- **Synthetic function of liver:** PT & albumin

### 4. IMAGING:

- USS
- MRCP/ERCP
- Liver biopsy (if ?malignancy)

# Gallbladder disease

→ Gallbladder **stores** and **concentrates** bile
→ Fatty acids / amino acids in duodenum stimulate **CCK** release
→ **CCK stimulates gallbladder contraction** & bile release

**Contents of bile:**

- Water (97%)
- Bile salts / pigments
- Cholesterol
- Phospholipids

## Investigations

1. **History & abdo exam** + pregnancy test + urine dip
2. **Bloods:** FBC, CRP/ESR, LFTs, clotting (± amylase)
3. **Imaging:**

→ USS (shows GB/duct dilation)
→ MRCP (95% sensitive to detect stone)
→ ERCP (Dx & Tx **but invasive)**
→ CT if concerned about tumour

USS = FIRST-LINE imaging

## Types of bile stone

1. **CHOLESTEROL** – crystallisation of **excess cholesterol**
2. **PIGMENT** – contain **calcium bilirubinate**
   - **Black:** haemolytic conditions (SCA, malaria)
   - **Brown:** biliary stasis or infection

| **Risk factors***: | |
|---|---|
| **F**at | **F**emale |
| **F**air | **F**ertile |
| **F**orty | **F**Hx |

*Crohn's & ileal resection = additional RFs as ↓ bile salt absorption

## Complications of gallstones

1. **In gallbladder:** biliary colic (cystic duct obstruction), acute cholecystitis, perforation*, carcinoma
   *needs emergency cholecystectomy
2. **In bile ducts:** biliary colic (common duct obstruction) → ascending cholangitis or acute pancreatitis
3. **In intestine:** gallstone ileus (stone erodes through gallbladder = fistula to duodenum → intestinal obstruction)
   AXR shows air in biliary tree

most gallstones = asymptomatic, until complications

**Mirizzi's syndrome:**

Gallstone impacts in **cystic duct / Hartmann's pouch**

- Extrinsic compression of hepatic duct
- **Obstructive jaundice** without dilation of CBD

## Management[4]

**OBSTRUCTION** → **INFLAMMATION** → **INFECTION** →

- *C. perfringens*
- *Klebsiella*
- *E. coli*

### BILIARY COLIC

= temporary duct obstruction
Systemically well!

**Symptoms:**

- **Colicky RUQ / diffuse pain** (visceral)
  - crescendos (stop if stone moves)
  - radiates to back/shoulder
  - worse with food (esp. fatty)
- **± N&V**
- **Obstructive jaundice**

if common duct blocked/Mirizzi's

**Investigations:**

- **Bloods = normal**
- USS/MRCP (shows dilation/stones)

**Management:**

- Bed rest, fluid, analgesia, NBM
- Elective **cholecystectomy** (by 6w)

### ACUTE CHOLECYSTITIS

= long-term *cystic duct* obstruction causes **inflammation of GB wall**
Systemically unwell!

**Symptoms:**

**Initially:** biliary colic

**With inflammation:** (localised pain)

- Fever & vomiting
- Severe RUQ pain + **peritonism**

**O/E:**

Guarding & rigidity + **Murphy's sign**

**Investigations:**

- **Bloods:** ↑ WCC, CRP, ESR
- USS/MRCP (dilation, thick GB wall)

**Management: ABCDE**

- IV fluid, analgesia + **IV ABX**
- Elective **cholecystectomy** (by 1w but ideally within 72h)

### ASCENDING CHOLANGITIS

= long-term *common bile duct* obstruction causes **duct infection**

**Symptoms:**

**Charcot's triad:**

1. High swinging **fever** (chills + rigors)
2. RUQ pain
3. Jaundice

**Reynolds' pentad:**
+ 4. Confusion
+ 5. Shock
(ICU admission)

**Investigations:**

- **Bloods:** ↑ WCC, CRP, ESR
- **LFTs:** obstructive picture

**Management: ABCDE + Sepsis 6**

- IV fluids, analgesia + **IV ABX** (e.g. cefuroxime + metronidazole)
- **Emergency ERCP** ± stenting

Key DDx: GORD, PUD, acute pancreatitis, IBD, acute hepatitis, pyelonephritis

Murphy's sign: press over GB – patient has sharp pain during inspiration as peritoneum hits your hand

---

[4] BMJ Best Practice (2021) *Gallstones, acute cholecystitis, acute cholangitis*

**Courvoisier's law:**

Painless, obstructive jaundice with palpable gallbladder = unlikely due to gallstones
→ think **carcinoma of pancreas**

## Other gallbladder pathologies

### CARCINOMA OF THE GALLBLADDER

→ Uncommon (occurs in elderly)
→ Associated with long-standing gallstones

**Symptoms:**

- RUQ pain
- Weight loss
- Obstructive jaundice & palpable mass (late sign)

} Sx occur late (& then mimic chronic cholecystitis)

**Management:** poor prognosis as late presentation

- Radical cholecystectomy (± liver resection if affected)

### CHOLANGIOCARCINOMA (adenocarcinoma of bile ducts / ampulla)

→ Common sites = at ductal confluences
→ Mostly in elderly patients
→ Can be 2° to PSC/IBD

**Symptoms:**

- Painless progressive jaundice
- Weight loss

} Sx occur late & mimic PANCREATIC CANCER

**Management:** poor prognosis as late presentation

- Whipple's procedure (if operable)
- Palliative stenting (relieves jaundice & helps gastric emptying)

# Pancreatic neoplasms

90% = **ductal adenocarcinomas** (60% head, 25% body, 15% tail). <2% = islet cell tumours (insulinoma, glucagonoma, gastrinoma). 95% involve a *KRAS2* mutation. Pancreatic neoplasms present late and are aggressive. They have a **poor prognosis**: 3% 5y survival, mean survival = 6m

**Risk factors for pancreatic neoplasms:**

- >80y
- Smoking/alcohol
- DM/obesity
- Chronic pancreatitis

## Symptoms

- **Painless obstructive jaundice** – pale stools, dark urine
- Weight loss, anorexia, **cachexia**
- Non-specific, dull, abdominal pain (may radiate to back)
- **New onset diabetes:** weight loss, polydipsia, polyuria (endocrine function)
- **Pancreatic enzyme insufficiency:** steatorrhoea (exocrine function)
- **Thrombophlebitis migrans (Trousseau's syndrome)** = thrombophlebitis following spontaneous clots

## Signs

- Courvoisier's sign
- Hepatosplenomegaly
- Epigastric mass
- Lymphadenopathy

**Courvoisier's law:**
A palpable gallbladder in the presence of **painless, obstructive jaundice** = unlikely due to gallstones

→ *think about a tumour in* ***head*** *of pancreas (presses on bile duct)*

## Investigations

- **Bloods:** FBC, U&Es, clotting, LFT (↑ conj. BR)
- **Tumour markers:** CA19-9* (level correlates with tumour volume)
- **Abdo USS:** pancreatic mass / dilated common bile duct, ascites
- **Endoscopic USS/ERCP & biopsy** (+ staging & grading)
- **Contrast-enhanced CT:** confirm Dx and staging

*CA19-9 = non-specific but can monitor progress

DIAGNOSTIC investigations

## Management

**Depends on tumour stage & patient factors**

Only 15–25% cure rate

1. **SURGICAL RESECTION** = Whipple's procedure (pancreaticoduodenectomy)
   - Resect head of pancreas, first part of duodenum, gallbladder & bile duct
   - **Complications:** delayed gastric emptying, bile leaks, pancreatic fistulae
2. **PALLIATIVE** = stenting & Sx relief ± chemo
   - Stents help jaundice & gastric outlet obstruction

Surgery only suitable if no mets and patient is fit

Post-op chemo can slow disease progression

# HAEMATOLOGY

# 05

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## ABBREVIATIONS

**Abs** – Antibodies
**AHA** – Autoimmune haemolytic anaemia
**ALL** – Acute lymphoblastic leukaemia
**AML** – Acute myeloid leukaemia
**APTT** – Activated partial thromboplastin time
**BM** – Bone marrow
**BMT** – Bone marrow transplant
**BR** – Bilirubin
**CF** – Clotting factor
**CLL** – Chronic lymphocytic leukaemia
**CML** – Chronic myeloid leukaemia
**CMV** – Cytomegalovirus
**CVL** – Central venous line
**CVS** – Chorionic villus sampling
**DIC** – Disseminated intravascular coagulation
**EPO** – Erythropoietin
**ET** – Essential thrombocythaemia
**Fe** – Iron
**FFP** – Fresh frozen plasma
**G6PD** – Glucose-6-phosphate dehydrogenase
**GCSF** – Granulocyte colony-stimulating factor
**Hb** – Haemoglobin
**HL** – Hodgkin lymphoma
**HPLC** – High performance liquid chromatography
**HS** – Hereditary spherocytosis
**HUS** – Haemolytic uraemic syndrome
**IDA** – Iron-deficiency anaemia
**ITP** – Immune thrombocytopenic purpura
**JGA** – Juxtaglomerular apparatus
**LDH** – Lactate dehydrogenase
**MAB** – Monoclonal antibodies
**MCV** – Mean corpuscular volume
**MGUS** – Monoclonal gammopathy of unknown significance
**MHC** – Mean haematocrit content
**MPN** – Myeloproliferative neoplasms
**NHL** – Non-Hodgkin lymphoma
**OGD** – Oesophago-gastro-duodenoscopy
**PK** – Pyruvate kinase
**Plt** – Platelets
**PMF** – Primary myelofibrosis
**PNH** – Paroxysmal nocturnal haemoglobinuria
**PRV** – Polycythaemia rubra vera
**PT** – Prothrombin time
**PVT** – Portal vein thrombosis
**RA** – Rheumatoid arthritis
**RBC** – Red blood cells
**Rh** – Rhesus
**SCD** – Sickle cell disease
**SLE** – Systemic lupus erythematosus
**TACO** – Transfusion-associated cardiovascular overload
**TIBC** – Total iron-binding capacity
**TTP** – Thrombotic thrombocytopenic purpura
**vWD** – von Willebrand disease
**WBC** – White blood cells

# Haematological lineage

## Definitions

**Haematopoiesis:** the production of blood cells and platelets
→ **Adults:** occurs in red bone marrow (vertebrae, ribs, pelvis)
→ **Children:** occurs in all bones
→ **Fetus:** occurs in liver & spleen

**Erythropoiesis:** RBC formation
→ Stimulated by EPO (produced by JGA in kidney in response to ↓$O_2$)
→ RBC lifespan = **120d**

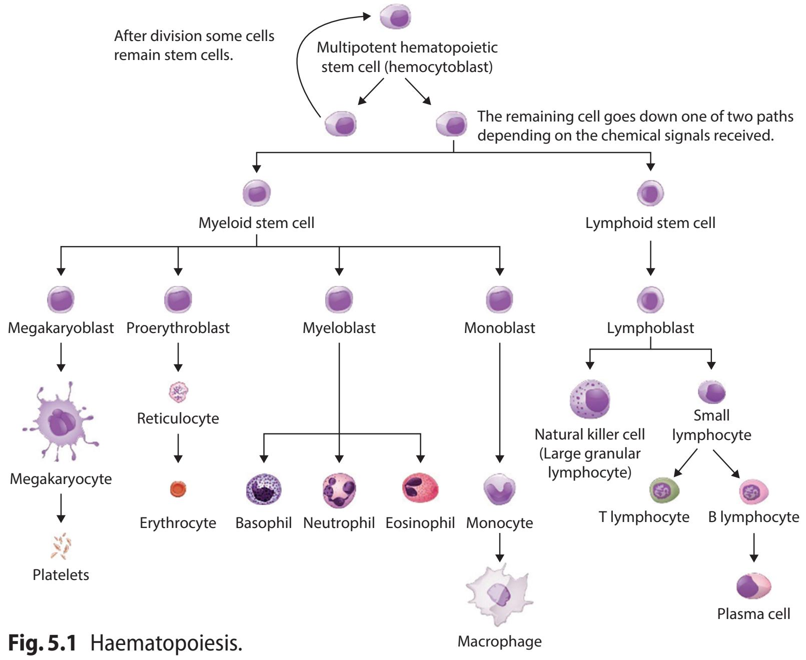
After division some cells remain stem cells.
Multipotent hematopoietic stem cell (hemocytoblast)
The remaining cell goes down one of two paths depending on the chemical signals received.
Myeloid stem cell
Lymphoid stem cell
Megakaryoblast
Proerythroblast
Myeloblast
Monoblast
Lymphoblast
Megakaryocyte
Reticulocyte
Natural killer cell (Large granular lymphocyte)
Small lymphocyte
Platelets
Erythrocyte
Basophil
Neutrophil
Eosinophil
Monocyte
T lymphocyte
B lymphocyte
Macrophage
Plasma cell

**Fig. 5.1** Haematopoiesis.

## Function of blood cells

**Red blood cells:** transport oxygen around the body (& $CO_2$ for removal)
**White blood cells:** form part of the immune system to fight infection
**Platelets:** involved in blood clotting

## Types of white cell

**Neutrophil** – chemotaxis & **phagocytosis**

**Lymphocyte** – **cell-mediated** immunity
→ **T cells** = regulate all parts of adaptive immune system
→ **B cells** = become plasma cells & secrete antibodies
→ **Natural killer cells** = pathogen destruction

**Eosinophil** – against **parasites** & **allergies**
**Basophil** – release **histamine** in inflammation
**Monocyte** – precursor of macrophages

**Agranulocytosis** = absences of circulating neutrophils
→ side-effect of carbimazole

| Neutrophilia (>10) | Neutropenia (<1.5) |
|---|---|
| • **Bacterial infection**<br>• **Corticosteroids**<br>• Neoplasia/**CML**<br>• Myeloproliferative disorder<br>• Bleeding/burns<br>• Inflammation<br>• Smoking<br>• GCSF | • **Viral infection**<br>• **Cytotoxic drugs**<br>• **Bone marrow failure**<br>• Severe sepsis<br>• Autoimmune (SLE)<br>• Hypersplenism (Felty's)<br>• B12/folate deficiency |

| Lymphocytosis (>5) | Lymphopenia (<1) |
|---|---|
| • **Viral infection** (EBV, CMV, HSV, VZV)<br>• **CLL & lymphoma**<br>• Chronic infection<br>• Septic shock / MI / trauma<br>• Smoking<br>• Raised BMI / metabolic syndrome | • **Bone marrow failure**<br>• **Cytotoxic drugs**<br>• **Bone marrow transplant**<br>• **Corticosteroids**<br>• CAR-T therapy<br>• Autoimmune (SLE)<br>• Renal failure<br>• HIV |

Purple points in tables = most important to be aware of

# Anaemia overview

Anaemia is defined as Hb <130g/L in men & <115g/L in women

## Causes

1. ↓ **RBC production** e.g. IDA (most common), BM disorders, cytotoxic drugs / chemotherapy, CKD, aplastic anaemia
2. ↑ **RBC destruction** e.g. SCD, thalassaemias, G6PD deficiency, autoimmune haemolysis
3. **Blood loss** (rare in children) e.g. vWD, Meckel diverticulum

## Signs/symptoms

### General signs/symptoms

- Fatigue/weakness
- Pallor (conjunctiva)
- SOB/tachycardia/dizziness

Symptoms occur if Hb <60–70g/L

### Specific signs/symptoms

→ **Koilonychia:** IDA
→ **Jaundice:** haemolytic anaemia
→ **Leg ulcers:** sickle cell disease
→ **Tingling fingers/toes:** B12 deficiency

## Investigations

- **FBC** – MCV (size of RBC) & MHC (Hb per RBC), reticulocytes
- **Iron studies** – serum iron & ferritin, TIBC
- **Blood film** – size, shape, colour of red cells
- **Serum bilirubin** – high in haemolysis
- **Hb HPLC** or **Hb electrophoresis** – shows amount of each Hb type (HbS, HbA, HbF)

**Types of haemoglobin**

**Fetal (HbF): 2α** chains + **2γ** chains → higher $O_2$ affinity
**Adult (HbA): 2α** chains + **2β** chains → lower $O_2$ affinity

*Adults normally have 2 HbA alleles (HbAA). Patients with haemoglobinopathies such as thalassaemia or SCD have at least one abnormal allele, causing non-HbA haemoglobins (e.g. HbSS, HbAS)*

**Key questions to ask:**

1. Isolated anaemia or pancytopenia? (pancytopenia suggests BM failure)
2. What is the MCV?

**All patients are screened for anaemia before surgery**

→ If **<100g/L** check haematinics and replace if low
→ If **<60g/L** as above, but transfuse if active bleeding

**DDx microcytic anaemia: TAILS**

- **T**halassaemia (α or β-thalassaemia trait)
- **A**naemia of chronic disease e.g. renal failure
- **I**ron-deficiency anaemia
- **L**ead poisoning
- **S**ideroblastic anaemia

**DDx macrocytic anaemia: ABCDEF**

- **A**lcohol & liver disease
- **B**12 deficiency
- **C**ompensatory reticulocytosis (blood loss)
- **D**rugs (cytotoxic)
- **E**ndocrine (hypothyroidism)
- **F**olate deficiency

**DDx normocytic anaemia: CHARMD**

- **C**hronic disease (↓ iron + ↑ ferritin)
- **H**aemolysis
- **A**cute blood loss
- **R**enal anaemia (causing low EPO levels)
- **M**arrow disorder
- **D**eficiencies combined (iron + B12)

# Anaemia from reduced RBC production

Must r/o GI bleed in older patients

## Iron-deficiency anaemia

→ ***most common anaemia worldwide*** *(especially menstruating females)*

**Causes:**

- **Inadequate intake** – Fe-deficient diet e.g. vegetarian
- **Malabsorption** – coeliac, gastrectomy
- **Increased requirements** – pregnancy
- **Chronic blood loss** – menorrhagia / GI bleed

NB. Ferritin will be raised in inflammation

**Diagnosis:**

1. **FBC:** ↓ MCV = microcytic
2. **Iron studies:** ↓ serum iron & ↓ serum ferritin, ↑ TIBC (body tries to ↑ Fe uptake)
3. **Blood film:** abnormally shaped, small, hypochromic (pale) RBCs

**Management[1]:**

1. **Determine cause:** thorough Hx & exam → r/o serious causes → e.g. GI malignancy
   - OGD/colonoscopy, anti-tTG, menorrhagia Hx
2. Treat underlying cause
3. Dietary advice
4. Oral iron supplements e.g. ferrous sulphate/fumarate
   - TDS until Hb normal then minimum 3m OD
   - Can use IV iron if oral not tolerated/absorbed

NB: if no response to Tx consider Ix for other causes (esp. malabsorption)

**Dietary advice for IDA**

**High Fe foods:**

- Red meat, liver
- Pulses, beans, peas
- Leafy, green veg
- Oily fish
- Fortified cereals
- Dried fruit / nuts

Vit C (fruit & veg) helps Fe absorption

**Foods to avoid:**

- Excess cow's milk (only 10% Fe is absorbed)
- Tannin (tea) (inhibits Fe absorption)

## B12 & folate deficiency

→ *causes pernicious anaemia*

**Causes of B12 deficiency:**

- **Low dietary intake** – vegan/vegetarian
- **Malabsorption** (in terminal ileum) – e.g. Crohn's, gastrectomy
- **Low intrinsic factor** – e.g. autoimmune (pernicious anaemia)

**Causes of folate deficiency:**

- **Low dietary intake**
- **Malabsorption** (in duodenum/jejunum) – e.g. coeliac, jejunal resection
- **Increased requirements** – pregnancy, haemolytic anaemia

**Diagnosis:**

1. **FBC:** ↑ MCV = macrocytic – often >125
2. **Blood film:** hypersegmented neutrophils (>5 lobules) & tear-drop cells
3. **Iron & B12 studies:** ↓ B12, ↓ serum folate, ↓ cobalamin
4. **Intrinsic factor antibodies**

**Management[2]:**

B12 = eggs, fortified cereals, dairy
Folate = broccoli, peas, brown rice

- If not pernicious anaemia, **dietary advice**
- IM B12 & folic acid (5mg OD) supplements (4m)
- If pernicious anaemia: **lifelong IM B12** replacement (hydroxocobalamin)

**Intrinsic factor** binds B12 in stomach to enable absorption in the terminal ileum

**B12** = coenzyme needed for folate conversion
**Folate** = needed for RBC synthesis

B12 deficiency can cause peripheral neuropathy, subacute spinal cord degeneration & angular cheilitis

**If B12 AND folate deficient, must replace B12 first to avoid subacute combined degeneration of the spinal cord**

## RBC aplasia

→ *failure of RBC synthesis*

**Causes:**

- **Diamond–Black anaemia** = rare, congenital → raised MCV ± short stature, abnormal thumbs
- **Transient erythroblastopenia** = triggered by viral infection in children
- **Parvovirus B19** – infects young RBCs – only causes RBC aplasia in children/ adults with inherited haemolytic anaemia

**Diagnostic results in RBC aplasia:** ↓ Hb, ↓ **reticulocytes,** normal BR, Coombs test –ve

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[1] BNF Treatment Summary – *Anaemia, Iron Deficiency*
[2] BNF Treatment Summary – *Anaemia, Megaloblastic*

# Haemolytic anaemias

## Clinical features

- Anaemia (normo-/macrocytic)
- Mild splenomegaly
- Jaundice

## Further investigations

- Hb electrophoresis
- Direct Coombs test

## Investigations

- **FBC:** ↓ Hb
- **Blood film:** ↑ reticulocytes* → lilac (polychromasia), strange shape
- **Bilirubin:** ↑ unconjugated BR
- **Lactate dehydrogenase:** ↑ LDH (freed from inside RBCs)
- **Haptoglobin:** ↓ (as binds to bilirubin)

*destruction of RBCs results in increased RBC production

## Extrinsic causes

### 1. AUTOIMMUNE HAEMOLYSIS (AHA) → autoantibodies cause extravascular haemolysis & spherocytosis

**Causes:**

→ **Primary**

→ **Secondary:** to another autoimmune condition, lymphoproliferative disorder or infection

Associated with Raynaud's
Can cause abdo pain and acrocyanosis

**Types:**

| | Warm AHA (direct Coombs IgA-positive) | Cold AHA (direct Coombs IgM-positive) |
|---|---|---|
| **Optimal binding** | 37°C | <4°C |
| **RBC destruction** | Extravascular (spleen/liver) | Intravascular (IgM binds complement) |
| **Management** | **Folate** plus:<br>**1st line:** steroids ± mycophenolate<br>**2nd line:** rituximab or splenectomy | **1st line:** folate and cold avoidance<br>**2nd line:** rituximab ± chemotherapy |

### 2. MICROANGIOPATHIC ANAEMIA → mechanical haemolysis triggered by physical trauma to RBCs in circulation by abnormal microcirculation with deposition of fibrin strands

**Causes:**

- Malignant HTN / pre-eclampsia
- HUS, DIC, vasculitis, TTP, post-BM transplant
- Prosthetic heart valve

**Investigations:**

- **FBC:** ↓ Hb, ↓ platelets
- **Blood film:** fragments
- **ADAMSTS13 level (cleaving enzyme):** low levels suggest TTP

**HUS/TTP:** normal coagulation
**DIC:** prolonged coagulation + ↓ fibrinogen

## Intrinsic causes

### 1. HEREDITARY SPHEROCYTOSIS (HS) = auto dominant or de novo mutations of RBC membrane proteins (1 in 5000)

ankyrin or spectrin

**Pathogenesis:** abnormal membrane can't deform well → can't pass through the spleen → **RBC destroyed in the spleen**

**Clinical presentation:** may present at birth, in children or adults

- **Jaundice** – may be intermittent
- **Anaemia** – mild (90g/L – normal) = tired, headaches
- **Splenomegaly** – due to RBC destruction in spleen
- **Gallstones** – persistent high bilirubin precipitates into stones
- **APLASTIC CRISIS*** – new RBCs not made fast enough

*usually children; follows parvovirus B19 & lasts 2–4w

**Diagnosis:** suspect if FHx and direct Coombs negative

1. **FBC & iron studies** – normocytic anaemia
2. **Blood film** – spherical RBCs with no central pallor + some microspherocytes

**Overview of causes**

| Intrinsic |
|---|
| **Hb alpha or beta chain abnormality** (thalassaemias, SCD)<br>**RBC membrane abnormality** (hereditary spherocytosis)<br>**Enzyme defects** (G6PD deficiency, pyruvate kinase deficiency) |
| **Extrinsic** |
| **Immune-mediated** → Coombs positive<br>→ Autoimmune (warm AHA & cold AHA)<br>→ Drugs (nitrofurantoin, penicillin, quinine) |
| **Non-immune mediated** → Coombs negative<br>→ Infection (malaria, CMV, *E. coli*, *Strep*.)<br>→ Microangiopathic |

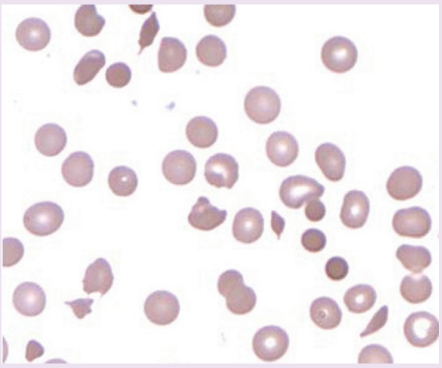

**Fig. 5.2** Spherical RBCs.

## 2. PYRUVATE KINASE (PK) DEFICIENCY = auto recessive mutation causes deficiency of PK enzyme

**Pathogenesis:** low levels of PK enzyme result in failure of RBCs to produce ATP → RBCs haemolysed prematurely

**Clinical presentation:** as for hereditary spherocytosis

- **Chronic haemolytic anaemia** – varies greatly in severity

**Diagnosis:**

1. **FBC & iron studies** – normocytic anaemia
2. **PK activity levels** – reduced (may not be if acute crisis)
3. **Direct Coombs test** – negative

**Management of HS and PK[3]:**

- **Oral folic acid** – ↑ need as ↑ RBC synthesis
- **Tx for aplastic crisis due to parvovirus:** transfusions until resolves (if chronic give IVIg)
- **Splenectomy** – if severe anaemia when young / poor growth / troublesome Sx
  → *will need daily prophylactic ABX afterwards*

## 3. G6PD DEFICIENCY = X-linked mutation in G6PD gene (enzyme involved in preventing oxidative damage to RBCs)

**Clinical presentation:** asymptomatic between haemolytic episodes

- **Neonatal jaundice** – usually within first 3d
- **Chronic haemolysis**
- **Acute intravascular haemolysis** – fever, pallor, malaise, dark urine
  → precipitated by infection, certain drugs & fava beans
  → Hb drops <50g/L within 24–48h

**Diagnosis:**

- **FBC:** ↓ Hb, ↑ reticulocytes
- **Unconjugated bilirubin:** raised
- **LDH:** raised
- **Blood film:** Heinz body inclusions* (lumps of damaged Hb)
- **G6PD activity:** reduced (may not be during an acute crisis – only check when stable)
- **Direct Coombs test** – negative

*only seen during haemolytic crisis; *ghost cells*

**Risk factors for G6PD deficiency:**
African, Mediterranean, Middle-Eastern ethnicity

**Management of G6PD deficiency:**

1. **Safety-net** for signs of acute haemolysis
2. **Advise** on foods/drugs to avoid (quinine, sulphonamides, nitrofurantoin, high dose aspirin, fava beans)
3. ± **Blood transfusion during haemolytic crisis**

## 4. PAROXYSMAL NOCTURNAL HAEMOGLOBINURIA (PNH)

**Pathogenesis:**

- A rare, acquired disorder of haematopoietic stem cells, which causes production of defective RBCs
- Defective cells are haemolysed by the body's complement system

**Symptoms:** range from mild → life-threatening

- Episodic haemoglobinuria (dark urine)
- Anaemia (SOB, chest pain, fatigue)

**Complications:**

- **Thromboses:** increased risk of blood clots (can result in PE, Budd–Chiari syndrome, organ ischaemia)
- **Pancytopenia:** increased risk of infection & bleeding + anaemia

**Management[4]:** depends on severity

1. **Symptomatic** e.g. iron replacement for anaemia
2. **Folate replacement** for ongoing haemolysis
3. ± **Anticoagulation:** thrombosis prophylaxis
4. ± **Monoclonal antibodies which block complement** – very successful

*Bone marrow transplant = only cure for PNH (reserved for most severe/life-threatening cases)*

## 5. THALASSAEMIAS (see overleaf)

## 6. SICKLE CELL DISEASE (see overleaf)

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[3] British Society for Haematology (2011) *Guidelines for the diagnosis and management of hereditary spherocytosis*

[4] NORD (2016) *The physician's guide to paroxysmal nocturnal haemoglobinuria (PNH)*

# Thalassaemias

Autosomal recessive disorders of alpha- or beta-globin production causing ↓ HbA

## Beta-thalassaemia

**There are 2 beta-globin genes, one for each beta chain**

→ **β-thalassaemia major:** both genes abnormal = no HbA = very severe anaemia

→ **β-thalassaemia minor:** 1 gene abnormal = almost normal Hb with marked low MCV (60s)

### CLINICAL FEATURES (BETA-THALASSAEMIA MAJOR):

- **Severe anaemia** from 3 to 6m old (when HbF runs out) → transfusion dependent
- Jaundice & pallor
- Hepatosplenomegaly if untreated
- Characteristic facies (frontal bossing) if untreated

### MANAGEMENT:

- **Lifelong monthly blood transfusions**
- Iron chelation (prevent Fe overload of heart & liver from transfusions)
- BM transplant = only cure

**Prognosis (major):**

90% live >40y if Tx-compliant

**Risk factors for beta-thalassaemia:**
Indian, Mediterranean, Middle-Eastern ethnicity

**Complications of long-term transfusion:**

1. **Fe deposition**
   - Heart = cardiomyopathy
   - Liver = cirrhosis
   - Pancreas = diabetes
   - Skin = hyperpigmentation
2. **Antibody formation (in 10%)**
3. **Venous access problems** – may need CVL

## Alpha-thalassaemia

**There are 4 alpha-globin genes, 2 for each chain**

→ **Deletion of all 4 α-globin genes:** α-thalassaemia major/ Hb Barts hydrops fetalis = death *in utero*

→ **Deletion of 3 α-globin genes:** HbH disease = mild/moderate anaemia (some HbA) → may need transfusions

→ **Deletion of 1–2 α-globin genes:** α-thalassaemia trait = asymptomatic (some HbA) → marked low MCV

**Risk factors for alpha-thalassaemia:**
South-East Asian ethnicity

### INVESTIGATING THALASSAEMIAS:

1. **FBC & iron studies** – microcytic, hypochromic anaemia
2. **Blood film:** 'target cells' or nucleated RBCs
3. **Hb HPLC** – proportions of HbA, HbF, $HbA_2$

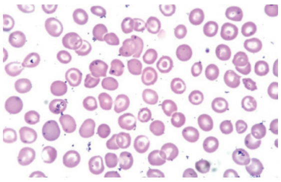

**Fig. 5.3** Blood film features of thalassaemia:
- target cells
- pale hypochromic cells

Diagnosis is confirmed with genetic testing

| | HbA | $HbA_2$ | HbF | HbS |
|---|---|---|---|---|
| **β-thalassaemia major** | X | ↑ | ↑ | X |
| **β-thalassaemia minor** | ✓ | ↑ | ↑↑ | X |
| **α-thalassaemia** | Normal | | | |
| **Sickle cell disease** | X | ✓ | ↑↑ | ✓ |
| **Sickle cell trait** | ✓ | ✓ | ↑↑ | ✓ |

$HbA_2$ = normal variant of Hb with levels higher than normal in thalassaemias

**Prenatal diagnosis (CVS):**
- offered if one/both parents affected
- 2 affected parents = 1 in 4 chance of each child being affected

# Sickle cell disease

= common genetic disorder in children → 1 in 2000 → autosomal recessive

## Cause

**Mutation in β-globin gene** which produces an **abnormal HbS chain** rather than HbA.

If low O$_2$, infection or acidosis, it polymerises and bends RBC into a sickle shape.

## Pathogenesis

Sickle-shaped RBCs have ↓ lifespan → get stuck & occlude vessels, causing infarction

| Type | Hb | Symptomatic? |
|---|---|---|
| **Sickle cell anaemia (HbSS)** | Homozygous for **abnormal HbS** | Yes (no HbA) |
| **HbSC disease (HbSC)** | 1 HbS gene + 1 **abnormal HbC** | Yes (less severe than SCA) |
| **Sickle β-thalassaemia (HbSA)** | 1 HbS + β-thalassaemia trait | Yes |
| **Sickle cell trait (HbSA)** | 1 HbS gene + 1 **normal HbA** | No (some HbA → carrier) |

## Clinical presentation

- **Moderate anaemia:** (60–100g/L)
- **Jaundice** (clinically detectable)
- **Infection** (↑ susceptibility especially pneumococcus & *H. influenzae*)

due to hyposplenism from chronic infarction

- **Splenomegaly** – due to acute sequestration (children only)
- **Painful vaso-occlusive crises:**
  - hands & feet – dactylitis & swelling
  - bones of limbs – avascular necrosis of femoral head
  - lungs – acute chest syndrome = EMERGENCY (Tx: CPAP + exchange transfusion)
  - penis – priapism
- **Acute anaemia (aplastic crisis):**
  - sudden ↓ Hb ± abdo pain ± hepatosplenomegaly

→ Triggers: infection, accumulation of sickle cells in spleen, parvovirus

## Diagnosis

1. **Screening:** pregnant women & neonates (Guthrie heel-prick test)
2. FBC & iron studies = ↓ Hb, low MCV, normal Fe
3. Blood film = sickle cells & target cells
4. LFTs & bilirubin = raised
5. Hb HPLC = HbS ± HbA
   ↳ diagnostic

asymptomatic for 6–8w after birth due to persistent HbF

## Management[5,6]

1. **Infection prophylaxis:** vaccines + daily PO penicillin lifelong (for hyposplenism)
2. **Daily folate supplementation** (lifelong)
3. **↓ risk vaso-occlusive crises:** dress warmly, stay hydrated, avoid excessive exercise/stress
4. **Treat vaso-occlusive crises:** PO/IV analgesia (morphine + ABX/O$_2$ if needed)
5. **Hydroxycarbamide to ↑ HbF:** if recurrent VO crises / acute chest
6. **Blood transfusion:** if acute complication (stroke, sepsis, aplastic crisis) & pre-operatively
7. **BM transplant:** only cure (for very severe cases)
8. **New treatments:** crizanlizumab → antibody binds P-selectin and stops RBCs sticking to endothelium

**Risk factors for SCD:**
Black, Afro-Caribbean ethnicity

**PROGNOSIS:** most severe form = 50% die by 40y from complications

**Long-term problems:**

- Short stature / delayed puberty
- Stroke & neuro damage
- Heart failure & renal dysfunction
- Pigment gallstones

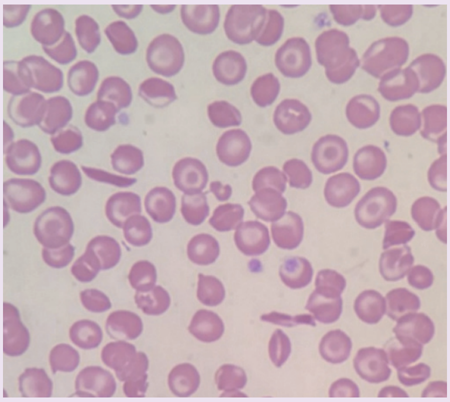

**Fig. 5.4** Sickle cells & target cells.

**Triggers of vaso-occlusive crises:** cold, infection, dehydration, hypoxia/altitude, drugs

**Management of vaso-occlusive crises[7]:**

1. ABCDE – IV fluids, O$_2$ only if sats <93%
2. **Analgesia***: IV/SC morphine **and** paracetamol/ibuprofen (co-prescribe laxatives & anti-emetics)
3. FBC, reticulocytes, infection screen
4. **Cross-match & transfusion**
5. Enoxaparin

*Most patients have a "pain plan"

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[5] British Society of Haematology (2018) *Guidelines for the use of hydroxycarbamide in children and adults with sickle cell disease*

[6] British Society of Haematology (2016) *Guidelines for transfusion in sickle cell disease part I & II*

[7] NICE (2012) *Sickle cell disease: managing acute painful episodes in hospital* [CG143]

# Pancytopenia

Combination of anaemia, leukopenia & thrombocytopenia

## Aetiology

1. Reduced blood cell production (bone marrow failure)
2. Increased blood cell destruction (immune-mediated)
3. Increased blood cell sequestration (in big spleen/liver)

## Differentials

| More common | Less common |
|---|---|
| • **Acute leukaemia**<br>• **Multiple myeloma**<br>• **Hypersplenism – cirrhosis, PVT, Felty's syndrome**<br>• Chemotherapy/radiotherapy<br>• B12/folate deficiency<br>• Some non-Hodgkin lymphoma<br>• Myelodysplasia | • Myelofibrosis<br>• Aplastic anaemia<br>• Haemophagocytic lymphohistiocytosis (HLH)<br>• Drug-induced<br>• Autoimmune (SLE, RA)<br>• Paroxysmal nocturnal haemoglobinuria<br>• Parvovirus in haemolytic anaemias<br>• Transfusion-related GVHD<br>• Hairy cell leukaemia<br>• Non-haematological malignancy (infiltrating bone marrow)<br>• Infection: HIV, hepatitis, CMV |

## Investigations

→ requires urgent haematology referral

1. **Thorough history** – PMHx, FHx, symptoms (bleeding, fatigue, recurrent infections, weight loss)
2. **Physical examination** – petechiae/purpura, lymphadenopathy, abdominal mass
3. **Blood tests** – FBC, retics, haemolysis screen, LFTs, clotting screen, B12, folate, HIV antibodies, serum electrophoresis
4. **Bone marrow aspirate & biopsy**
5. **Imaging** – CT or PET if lymphoma

## Management

**Immediate:** RBC + platelet transfusion ± antibiotics
**Further management:** determine and treat underlying cause

# Myeloproliferative disorders

Bone marrow disorders with **differentiated myeloid cell subtype expansion** in blood (e.g. Hb, platelets or white cells). Known as **myeloproliferative NEOPLASMS** (MPNs)

**Complications:**

- **Thrombosis**
- Bleeding
- Evolution (to AML or 2° myelofibrosis)

## Investigations

- Blood tests (FBC, LFTs)
- Bone marrow biopsy for CML and primary myelofibrosis (PMF)
- Genetic tests
  → must rule out *BCR–ABL* fusion gene (suggesting CML), before diagnosing any other MPN

## Types

| Disorder | Cell type affected / increased | Symptoms | Treatment | Prognosis |
|---|---|---|---|---|
| **Polycythaemia rubra vera** | • **RBCs high**<br>• Plts normal or ↑<br>Associated *JAK2* mutation | • Headaches<br>• Fatigue<br>• Blurred vision<br>• Pruritus<br>• HTN<br>• Sx due to blood clots | **Reduce clot risk:**<br>Venesection + low dose aspirin (tablet chemotherapy if platelets also high) | Normal life expectancy<br>**5% develop AML** |
| **Essential thrombocythaemia** | • **Platelets high**<br>• Rest normal<br>Associated JAK2 or *CAL-R* mutation | • Abdominal pain<br>• Mottled skin<br>• Stroke<br>• Burning hands/feet (erythromelalgia) | **Reduce stroke risk:**<br>Low dose aspirin (tablet chemotherapy if >60y & platelets high) | Normal life expectancy<br>**5% develop AML** |
| **Chronic myeloid leukaemia** | • **WBCs very high (basophilia)**<br>• Plts usually high<br>• Anaemia<br>t(9,22) mutation causing *BCR–ABL* fusion gene (Philadelphia chr) | • Infection<br>• Fatigue<br>• Bruises/bleeds<br>• Bone pain<br>• Abdominal pain (splenomegaly)<br>• Night sweats<br>• Weight loss | **Targeted therapy:**<br>Tablets which target the *BCR–ABL* gene → effectively 'cures' most patients<br>**Bone marrow transplant** if targeted Tx unsuccessful | Mostly normal life expectancy with targeted therapy<br>**Very few develop AML** |
| **Primary myelofibrosis** | • **Scar tissue**<br>(prevents cell production) | • Fatigue<br>• Shortness of breath<br>• Bruises/bleeds<br>• Abdominal pain (splenomegaly)<br>• Bone pain<br>• Night sweats<br>• Weight loss<br>• Gout | 1. Blood transfusions / EPO<br>2. Low dose aspirin if ↑ plts<br>3. Allopurinol for gout<br>4. *JAK2* inhibitors if *JAK2* mutation<br>5. Chemotherapy + stem cell transplant if young | Depends on degree of low blood counts<br>Some may only live months<br>**10–20% develop AML** |

# Multiple myeloma

Malignant clonal proliferation of **plasma cells** (due to B cell mutations) → excess secretion on one type of paraprotein / light chain (e.g. IgG, IgA). Median age = 70y

## Clinical presentation

| | | |
|---|---|---|
| **C** | Hyper**C**alcaemia | ↑ osteoclast activity → fluids + bisphosphonates |
| **R** | **R**enal dysfunction | Deposition of light chain Ig → fluids + dexamethasone |
| **A** | **A**naemia | BM crowding suppresses erythropoiesis |
| **B** | **B**leeding | BM crowding suppresses platelets |
| **B** | **B**one pain/lesions | ↑ osteoclast activity → bisphosphonates + surgery |
| **I** | **I**nfection | Reduction in normal Igs → vaccinations, CSGF |

## Investigations

**1. BLOODS:**
- FBC (↓ Hb, ↓ WCC, ↓ plts)
- ESR (raised)
- U&Es (deranged in 20%)

**2. BONE PROFILE**
- Ca (raised)
- ALP (normal)

**3. SERUM ELECTROPHORESIS**
- Serum free light chains / paraprotein (IgG/IgA)

**4. X-RAY:**
- Lytic lesions (‘pepper-pot’)

**5. MRI/PET SCAN:**
- Lytic lesions

**6. BM BIOPSY**
- >10% plasma cells = myeloma
- <10% plasma cells = MGUS

## Management

**Mx = palliative (but many now have >5y prognosis)**

**Several lines of chemo + immuno-modulatory agents**

+ bone protection with bisphosphonates
+ infection prophylaxis (vaccinations, antiviral)
+ radiotherapy for pain in isolated sites

→ Bone marrow transplant if fit (to prolong remission)

Green text denotes treatment options

**Emergency presentations:**

- Pathological vertebral fracture
- Bone pain
- Acute renal failure
- Spinal cord compression
- Hyperviscosity
- Hypercalcaemia

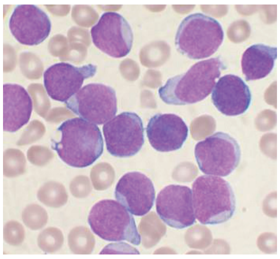
= lethargy, constipation, thirst, nausea, polyuria

# Leukaemia

**Poor prognostic factors of ALL:**
**Age** <1 or >10y
**WBC** $>50 \times 10^9/L$
**CNS involvement**
**Cytogenic abnormalities in tumour cells**
**Persisting blast cells** after initial chemo

Cancer of the white blood cells

## Acute leukaemias

| | Acute lymphoblastic (ALL) | Acute myeloid (AML) |
|---|---|---|
| **Pathogenesis** | Rapidly proliferating lymphocyte stem cells = accumulation of **immature lymphoblast cells** (can be B-cell or T-cell) | Malignancy of myeloid stem cells = accumulation of **immature myeloblasts** |
| **Peak age** | 2–5y<br>(80% of childhood leukaemias) | >60y |
| **Symptoms** | **Rapid onset (1–2w)**<br>May see mediastinal mass in T-cell ALL<br>• **Anaemia:** lethargy, pallor, SOB<br>• **Neutropenia:** sore throat, recurrent fevers, infections<br>• **Thrombocytopenia:** bruising, petechiae, nose bleeds<br>• **End organ infiltration:** lymphadenopathy, splenomegaly<br>• **General:** fatigue, malaise, anorexia, **bone pain** | |
| **WCC** | **May be high** (immature blast cells) or **may have pancytopenia** | |
| **Investigations** | 1. **FBC:** ↓ Hb, ↓ plts, ↓ neutrophils, ↑ or ↓ WCC<br>2. **Peripheral blood film:** blast cells (± Auer rods in AML)<br>3. **Clotting screen:** may have DIC<br>4. **Bone marrow biopsy → diagnostic & prognostic** (gene mutations)<br>5. **CXR:** mediastinal mass (characteristic of T-cell ALL)<br><br>**Fig. 5.5** Blast cells. | |
| **Management** | 1. **Pre-chemo:** dexamethasone (↓ lymphoblasts)<br>2. **Chemotherapy:** induce* → intensify → maintain (2–3y maintenance)<br>3. **Concurrent treatment:** intrathecal chemo (MTX) | 1. **Pre-chemo:** hydroxyurea (lower WCC)<br>2. **Intensive chemotherapy:** 3–4 cycles<br>3. **Risk stratification for further Tx**<br>→ Further chemotherapy<br>→ Stem cell transplant |
| **Prognosis** | **Children:** 90% cure rate<br>**Adults:** 35–40% 5y survival | Depends on age, genetics and response |

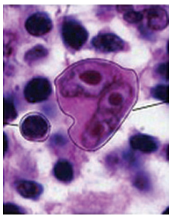
***Risk stratification:**
- after induction to decide next step
- adults often need BM transplant
- children almost never need BM transplant

## Chronic leukaemias

| | **Chronic lymphocytic (CLL)** | **Chronic myeloid (CML)** |
|---|---|---|
| **Pathogenesis** | Accumulation of **apoptosis-resistant B cells** | Increased production of myeloid progenitors<br>→ 95% have **Philadelphia chromosome (9:22)** |
| **Peak age** | 70y M>F = 2:1<br>(most common adult leukaemia) | 40–60y |
| **Symptoms** | **Often asymptomatic** (usually incidental finding)<br>• **Anaemia:** lethargy, pallor, SOB<br>• **Neutropenia:** recurrent infections<br>• **Thrombocytopenia:** bruising, petechiae<br>• **B symptoms:** malaise, night sweats, weight loss | **Insidious onset** (non-specific symptoms)<br>→ **no bone marrow failure**<br>• **General:** tiredness/lethargy<br>• **Massive splenomegaly** (abdominal discomfort) |
| **WCC** | **WCC normal to very high** (mature lymphocytes) | **Very high WCC** (neutrophils, eosinophils & basophils*)<br>*the only cause of significant basophilia |
| **Investigations** | 1. **FBC:** ↓ Hb, ↓ plts, ↑ WCC, ↑ lymphocytes (if advanced disease)<br>2. **Peripheral blood film:** smudge cells<br>3. **Flow cytometry** on blood to identify CLL cells<br>4. **Bone marrow biopsy**<br>→ to look for prognostic genetic markers | 1. **FBC:** ↑ plts, ↑↑↑ WCC, ↑ neutrophils, ↑ basophils<br>2. **Peripheral blood film:** mature myeloid cells<br>3. **BM biopsy → diagnostic & prognostic**<br>4. **Cytogenetics:** blood/BM (Philadelphia Chr)<br>5. **Molecular assessment:** mutation t(9,22) (*BCR–ABL* fusion gene) |
| **Management** | **If no symptoms:** watch & wait<br>**If B symptoms / low Hb / low platelets:**<br>1. **Chemotherapy:** more intensive if younger<br>2. **Targeted therapy:** BTK inhibitors, BCL2 inhibitors, monoclonal antibodies | 1. **Tyrosine kinase inhibitors (TKI): imatinib**<br>→ Monitor response<br>→ Some develop resistance to TKIs & need BMT |
| **Prognosis** | **85% 5y survival**<br>A few transform into aggressive lymphoma = very bad prognosis | **70% 5y survival**<br>Without treatment transform into AML = very bad prognosis |

# Lymphoma

→ **solid malignancies of immune system**
→ due to lymphocyte proliferation in LNs, spleen, thymus, BM
→ about 70 different types

| | **Hodgkin lymphoma (10%)**<br>Reed–Sternberg cells | **Non-Hodgkin lymphoma (90%)**<br>no Reed–Sternberg cells |
|---|---|---|
| **Aetiology** | Reed–Sternberg cells (mutated B cell)<br>→ 50% associated with EBV (usually if older patients) | → More common in patients on immunosuppression or with autoimmune disease<br>**High grade:** rapid & aggressive<br>→ curable but if relapse then poor prognosis<br>**Low grade:** slow growing<br>→ not generally curable but very good long-term prognosis as many effective treatments |
| **Peak age** | **Bimodal:** 15–35y **and** 60y | 70y |
| **Symptoms** | • **Painless lymphadenopathy** (usually **cervical**)<br>► may be **mass effects** (SVC/bronchi obstruction)<br>► becomes painful with alcohol<br>• B symptoms (in 40%) | • **Painless lymphadenopathy** (cervical or **abdo**)<br>► often cause mass effects<br>• Abdo symptoms (mass/pain)<br>• B symptoms (in 20%) |
| **Investigations** | • FBC & blood film<br>• Lymph node biopsy/excision = **diagnostic** (Reed–Sternberg cells in HL)<br>• Bone marrow biopsy<br>• CT or FDG-PET – stage & grade<br><br>**Fig. 5.6**<br>Reed–Sternberg cells. | |
| **Management** | **B-cell:** immunochemotherapy ± radiotherapy<br>**T-cell:** depends on type → poorer prognosis<br>Immunochemotherapy (chemo + MAB) or just chemotherapy<br>• **Low grade:** follicular, marginal, mantle zone<br>► Watch & wait if asymptomatic<br>► OR multi-agent chemo + monoclonal Abs<br>► Radiotherapy if stage 1 disease<br>• **High grade:** Burkitt / diffuse large B-cell<br>► Multi-agent chemo + monoclonal Abs<br>► ± radiotherapy<br>**New treatments:** bispecific antibodies and CAR-T (GM modified T cells which target cancer) | |

# Haemostasis

## Primary haemostasis

→ *platelet-mediated (plts made in BM)*

1. **Vasoconstriction** – slows blood for plt adhesion
2. **Platelet Adhesion** then Activation then Aggregation (3 As)
3. **Haemostatic plug** – platelets + fibrinogen = soft 1° **plug**

→ Plug = short-lived & easily sheared off

### CLINICAL PRESENTATION OF IMPAIRED 1° HAEMOSTASIS

- **Mucocutaneous bleeds:** bruising, petechiae, epistaxis, menorrhagia, gum bleeds
- **Intra-op / immediate post-op bleeding**

### CAUSES OF IMPAIRED 1° HAEMOSTASIS

1. **↓/ impaired vWF** (vWF disease) = platelets can't bind endothelium
2. **Impaired platelet function**
    - Congenital – Bernard–Soulier, Glanzmann's
    - Medications – antiplatelet drugs
    - Other – uraemia, myeloproliferative disorders
3. **Reduced platelet number:** thrombocytopenia

(<150, but problems unlikely if >30, unless trauma or surgery)

***Key causes**

| Causes of thrombocytopenia | | |
|---|---|---|
| **Increased destruction** | **Non-immune:**<br>• Sepsis, DIC, TTP | |
| | **Immune:**<br>• ITP<br>• Post-transfusion<br>• Drugs (NSAIDs, heparin, vancomycin, quinine) | |
| **Decreased production** | • Alcohol<br>• Cytotoxic drugs<br>• Leukaemia / aplastic anaemia / myelodysplasia<br>• BM malignancy<br>• Infections | |
| **Other** | • Congenital<br>• Hypersplenism<br>• Haemodilution | |

## Secondary haemostasis

→ *clotting factor mediated (CFs made in the liver)*

→ After initial platelet aggregation, the platelet membrane 'flips' & provides a surface for binding of coagulation factors

→ This **stabilises the fibrinogen plug**

→ Activation of **clotting cascade:** chain reaction resulting in cleaving of **fibrinogen → fibrin**

### CLINICAL PRESENTATION OF IMPAIRED 2° HAEMOSTASIS

- **Muscular/soft tissue bleeds:** extensive bruising
- **Haemarthrosis**
- **Delayed post-op bleeding** & poor wound healing

### CAUSES OF IMPAIRED 2° HAEMOSTASIS

**Reduced coagulation factors:**

- Congenital: haemophilia
- Acquired: anticoagulant drugs, dilution post-transfusion, liver dysfunction, DIC

**Mechanism of plug formation:**

→ **Vessel wall damage** exposes vWF & collagen

→ **Platelet glycoproteins bind vWF** & collagen which activates platelets and allows binding to fibrinogen

→ **Soft haemostatic plug** forms

**Antiplatelet drugs:**

1. **Aspirin & NSAIDs:**
   irreversible COX inhibition
   = ↓ PGs & TXA = ↓ plt aggregation
2. **Clopidogrel & ticagrelor:**
   inhibits ADP binding to platelet GP receptors
   = no platelet activation or binding
3. **ABCIXIMAB/eptifibatide:**
   competitively bind GP2B/3A receptor
   = inhibits plt binding & activation

**Indications:**
1° or 2° prevention of atherothrombotic events (e.g. MI in angina, stroke in AF)
2° prevention of cardiovascular event post MI/stroke

**Risks:** ↑ bleed risk (risk of GI & intracerebral bleeds)

**IMMUNE THROMBOCYTOPENIA (ITP)**
**= autoantibodies against platelets**

**Result:** isolated thrombocytopenia with **no other clear cause**

**Presentation:** acute, persistent (>3m) or chronic (>12m) features of 1° dysfunction → often purpura, bleeding gums

**Treatment:** steroids ± IVIg
(2nd line = rituximab, splenectomy, immunosuppressants)

**Vit K dependent clotting factors:**
2, 7, 9, 10

# Coagulation disorders

## Disseminated intravascular coagulation

**Definition:** acquired syndrome with inappropriate systemic activation of coagulation pathways

### PATHOGENESIS:

Consumption of CFs & plts → if severe can cause organ dysfunction, infarction, and bleeding

### CAUSES:

Infection, trauma/burns, malignancy, severe liver failure, obstetric complications, ABO mismatch transfusion

### PRESENTATION:

Bruising, excessive bleeding, renal failure (AKI due to hypoperfusion)

### INVESTIGATIONS:

- FBC = low plts
- Coag screen = ↑ PT & APTT
- ↑ D-dimers & ↓ fibrinogen

### MANAGEMENT:

- Aggressive resuscitation
- Treat cause
- Support with FFP/cryoprecipitate only if active bleeding

## Haemophilia

**Haemophilia A:** factor VIII deficiency
(1 in 5000)
**Haemophilia B:** factor IX deficiency
(1 in 30,000)

**Definition:** X-linked recessive coagulation disorder

Nearly always males

### CLINICAL FEATURES:

**Mild:** bleed after surgery

**Moderate:** bleed after minor trauma

**Severe:** spontaneous joint/muscle bleeds (haemarthrosis)

**complication = arthritis**

→ **40% present in neonates:** (with intracranial haemorrhage / post circumcision bleed)

→ **Commonly present when start to crawl/walk**

**Severity based on amount of clotting factor:**

**Mild:** >5%
**Mod:** 1–5%
**Severe:** <1%

### INVESTIGATIONS:

- **FBC, blood film, LFTs**
- **Coag screen:** ↑ APTT & **normal bleed time**
- **CF assays:** low factor VIII or IX

### MANAGEMENT: → avoid NSAIDs & IM injections

- **For any acute bleeds:**
  - Elevation, compression & tranexamic acid
  - IV recombinant factor VIII/IX → given ASAP (parents taught how to administer at home)
- **For mild disease:** desmopressin (DDAVP) = antidiuretic that causes secretion of factor VIII & vWF into plasma

  Risk of hyponatraemia
- **For severe disease or before major surgery:** regular prophylactic IV factor

A new monoclonal antibody Tx now available that is longer-acting: binds factor IXa and factor X → mimics activity of factor VIII

## von Willebrand disease

**Definition:** autosomal dominant deficiency/defect in von Willebrand factor (vWF)

### CLINICAL FEATURES:

- Bruising
- Prolonged bleeding
- **Mucosal bleeding** – epistaxis, menorrhagia

### DIAGNOSIS:

Mild ↑ APTT, **normal platelets & INR**
= Prolonged bleed time

*Most common inherited bleeding disorder*

### MANAGEMENT OF BLEEDS: → avoid NSAIDs & IM injections

**For any acute bleeds:** elevation, compression & tranexamic acid

- **Mild (10–50%):** PO/IV tranexamic acid ± SC or nasal desmopressin (DDAVP)
- **Moderate/severe:** recombinant vWF

**von Willebrand factor:**

- facilitates platelet adhesion
- carrier protein for factor VIII preventing its degradation

## Thrombophilias

**Definition:** acquired or inherited deficiency/defect in natural anticoagulants → predisposes to thrombosis

### CAUSES:

1. **Inherited:**
    - Factor V Leiden and prothrombin mutations (most common in north Europe)
    - Protein C/S deficiency
2. **Acquired:**
    - Antiphospholipid syndrome

### THROMBOPHILIA SCREEN: (most hospitals have guidelines)

- FBC & clotting
- Protein C & S levels, homocysteine
- Antiphospholipid Abs (lupus anticoagulant, anticardiolipin, B2-glycoprotein antibodies)
- ± Factor V Leiden and prothrombin mutations

**Consider thrombophilia screen if:**

- Arterial thrombosis <50y
- Venous thrombosis <40y & no RFs
- Recurrent, unexplained VTE
- Familial VTE
- Recurrent miscarriage
- Unusual site of thrombosis e.g. mesenteric veins

# Investigating bleeding problems

## History & examination

- **Symptoms:** bleeding/bruising/menorrhagia; where, how much, associated Sx
- **PMHx/FHx:** bleeding/clotting disorders, liver dysfunction, autoimmune
- **DHx:** anticoagulants, NSAIDs, ABX
- **SHx:** alcohol, smoking

Ask if ever happened before
e.g. with operations/dental work

**Performing clotting studies:**
**Blue-topped bottle**
(2nd in draw order after blood cultures)

## Coagulation testing

### PRIMARY HAEMOSTASIS

1. **FBC & blood film:** plt number & morphology = **key tests**
2. **Other:** vWF assays, plt func. (not routine)

### SECONDARY HAEMOSTASIS

1. **Clotting screen**
    - **PT** = extrinsic pathway (CF 7 & 10)
    - **APTT** = intrinsic pathway (CF 8-12)
    - **Thrombin time** = time for fibrinogen to make fibrin

**↑ in both PT & APTT =**
**a)** common path problem (CF II, V, X) **OR**
**b)** global CF deficiency (e.g. vit K antagonism affecting CF II, VII, IX, X)

2. **Mixing studies:** patient plasma + FFP
    - Full correction indicates CF deficiency
    - No correction indicates CF inhibition (i.e. antibody against CF)
3. **Fibrinogen** and fibrinogen degradation products / D-dimers
4. **CF assays:** measure individual CF levels (depending on mixing studies / other tests)

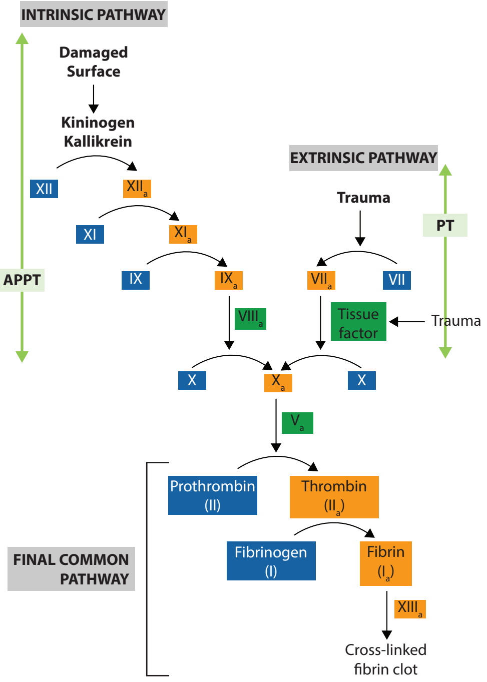

INTRINSIC PATHWAY
Damaged Surface
Kininogen Kallikrein
XII
$XII_a$
XI
$XI_a$
IX
$IX_a$
$VIII_a$
APPT
EXTRINSIC PATHWAY
Trauma
PT
$VII_a$
VII
Tissue factor
Trauma
X
$X_a$
X
$V_a$
Prothrombin (II)
Thrombin ($II_a$)
FINAL COMMON PATHWAY
Fibrinogen (I)
Fibrin ($I_a$)
$XIII_a$
Cross-linked fibrin clot

**Fig. 5.7** Coagulation cascade.

**Types of anticoagulant**

**Heparins:** act on CFs II & X – cause **prolonged APTT → reverse with protamine sulphate (or just wait as short half-life)**

**Warfarin:** vitamin K antagonist affects CFs II, VII, IX, X to cause **prolonged APTT & PT → reverse with vit K / prothrombin complex**

**DOACs:** reversible inhibitors of CF Xa → **NB: they have poor correlation with APTT & PT → reverse with specific inhibitors**

# Blood transfusion

## Blood groups

- Blood group determined by **antigens on RBCs** → encoded by A, B & O genes on chromosome 9
- **From 3 months old** we produce natural **IgM antibodies** against the A or B antigens that we don't have
- Receiving **ABO incompatible blood** can cause **massive transfusion reaction & death**

| Group | UK prevalence | Antigens | Antibodies |
|---|---|---|---|
| **O** | 47% | None<br>Universal donor | A & B |
| **A** | 42% | A | B |
| **B** | 8% | B | A |
| **AB** | 3% | A & B | None<br>Universal recipient |

Universal donor blood is O Rh –ve

**Alleles / inheritance of blood group:**
**O** = OO → recessive
**A** = AA, AO → co-dominant
**B** = BB, BO → co-dominant
**AB** = AB → co-dominant

## Rhesus antigen

- Encoded by 2 genes (D gene & CcEe gene)
    - **D** = transcribed = Rh +ve
    - **d** = not transcribed = Rh –ve
- Rh –ve individuals will produce **IgG antibodies (anti-D)** if exposed to d-antigen
  → via **transfusion** or **pregnancy with D+ fetus**
  → can cause haemolytic disease of newborn if mother Rh –ve and fetus Rh +ve

**1 unit blood increases Hb by 10g/L**
(repeat Hb measurement 30min post-transfusion)

Transfuse 1U over 2h if non-urgent

## Blood products

| Product | Indication |
|---|---|
| **RBC** | Acute anaemia **<70g/dL***, blood loss (haemorrhage), bone marrow failure, SCD/thalassaemia |
| **Platelets** | Bleeding or Plt count $<10 \times 10^9$ due to DIC with bleeding or chemotherapy → **must not give if ITP or TTP** |
| **FFP** | Clotting defects (e.g. DIC, liver disease, TTP), major haemorrhage |
| **Cryoprecipitate** | Fibrinogen / factor VIII replacement (e.g. haemophilia, vWD, DIC with bleeding) |
| **Human albumin solution** | Protein replacement (e.g. ascitic tap) |

**Alternatives to blood transfusion:**

- Cell savers (recycle & clean patient's own blood during operations)
- Iron infusion (if iron-deficient) or B12/folate replacement (if folate-deficient)
- EPO

***Low Hb does not necessitate transfusion**
→ establish cause; transfuse if severe symptoms e.g. chest pain / syncope
*Always consider transfusion in haemorrhage (e.g. melaena)

Do not transfuse patients with very low B12 or folate

## Process of blood transfusion[8]

→ *only transfuse if no suitable alternative*

### PRE-COLLECTION OF BLOOD PRODUCTS:

- **CAREFULLY LABEL** samples for cross-match to ensure no mistakes
- **Explain risks and benefits** to patient & **document their verbal consent** → give patient info leaflet
- Assess & **document TACO risk** in patient
- Complete prescription **including special requests****
- Go through pre-collection checklist incl. any previous transfusion reactions
- Prep patient: record obs & insert wide-bore cannula

****Request irradiated blood if:**
- Haematological malignancy
- Aplastic anaemia
- Hx of bone marrow transplant
- Immunodeficiency syndrome
- On some chemotherapy

---

[8] British Society for Haematology (2017) *The administration of blood components*

**PRE-TRANSFUSION CHECKS:** **must be done by 2 people in some hospitals**

- Patient ID & consent
- Component prescription – correct type, compatibility, correct rate, signed by doctor
- Expiry date & appearance of unit – any discolouration/damage
- Document date & time of start → complete & stick pink sticker on prescription chart & return blue sticker to blood bank

**POST-TRANSFUSION:** → **record obs 15 mins after starting each unit** (temp., pulse, RR, BP)

## Massive blood transfusion

→ *replacement of 10 units / entire blood volume within 24h*

**Indications:** trauma, ruptured AAA, GI bleed, cardiac surgery etc.
**Complications:** dilution of platelets & clotting factors → **triad:** acidosis, hypothermia, coagulopathy
**Major transfusion protocol:** triggered **if SBP <80mm/Hg** → combine RBCs with FFP & cryoprecipitate

**Temp ≥39°C or ↑ ≥2** from baseline = **stop transfusion** & senior r/v

## Complications of blood transfusion

→ *report immediately to haematology / blood bank & complete Datix*

### 1. HAEMOLYTIC TRANSFUSION REACTION:

**Pathogenesis:** mismatched ABO group = complement activation & intravascular haemolysis
**Symptoms:** ↑ temp, ↓ BP, ↑ HR, abdo/chest pain, anxiety
**Complications:** shock, renal failure, DIC & death
**Management:** **stop transfusion** → ABCDE, saline, check identity of unit
*** Tell blood bank, repeat G&S sample, do haemolysis and coag screen, and send this with blood unit to blood bank ***

### 2. NON-HAEMOLYTIC FEBRILE REACTION:

**Symptoms:** shivering & fever 1.5h post-transfusion
**Management:** slow/stop transfusion + antipyretic

### 3. ALLERGY/ANAPHYLAXIS:

**Mild allergy:** urticaria, itch

- Mx = slow transfusion + chlorphenamine

**Severe anaphylaxis:** bronchospasm, wheeze/stridor, cyanosis

- Mx = **stop transfusion**, ABCDE/anaphylaxis Tx

*** Tell blood bank and check IgA (may be IgA-deficient and so have made antibodies to IgA = 1 in 700) ***

### 4. BACTERIAL CONTAMINATION:

More common with platelet transfusion

**Symptoms:** rigors, fever, hypotension → shock
**Management:** **stop transfusion** & start Sepsis 6
*** Tell blood bank & culture the blood unit ***

### 5. TRANSFUSION-ASSOCIATED LUNG INJURY (TRALI):

**Pathogenesis:** due to Abs in donor plasma vs. host neutrophils, FFP or platelets (FFP & platelets contain more antibodies so greater risk of reaction)
**Symptoms:** SOB, cough, hypoxia, white-out on CXR (2–6h post-transfusion)
**Management:** **stop transfusion**, ABCDE, 100% $O_2$
*** Tell blood bank and screen the donor unit for antibodies → they may have donated other units ***

### 6. TRANSFUSION-ASSOCIATED CIRCULATORY OVERLOAD (TACO):

**Symptoms:** within 6h post-transfusion
- Dyspnoea, hypoxia
- Tachycardia, hypertension
- Basal crepitations (pulm. oedema)

**Management:**
- Slow/stop transfusion
- $O_2$ – may need ventilation
- Consider exchange transfusion

**Risk factors for TACO:**
- >70y
- Low weight / child
- Cardiac failure
- Renal impairment / low albumin

### 7. TRANSFUSION-ASSOCIATED GRAFT VS. HOST DISEASE (TA-GVHD) → rare

→ occurs 1–3w post-transfusion

**Pathogenesis:**
- Only seen in immunosuppressed, as normal immune systems kill lymphocytes transfused in units of blood
- In TA-GVHD, lymphocytes from the transfused blood attack the recipient's skin, bone marrow, and gut

**Symptoms:** rash, fever, diarrhoea, BM failure 1–3w post-transfusion

**Need to give irradiated blood to at-risk patients** (see above)

### 8. DELAYED HAEMOLYTIC TRANSFUSION REACTION

→ occurs 24h to 30d post-transfusion

**Pathogenesis:**
- Slower antibody response to a foreign antigen on transfused blood than with ABO mismatch
- Causes extravascular haemolysis, and progressive anaemia

**Symptoms:** fever, haemolysis (jaundice), dark urine & falling Hb

**Tests:**
1. **Haemolysis screen:** ↑ LDH, ↑ unconjugated bilirubin, ↓ haptoglobin
2. **Direct Coombs test:** positive
3. **Antibody identification** – to ensure patient doesn't receive a unit expressing that antigen again

**Treatment:** symptomatic Tx (paracetamol) + avoid future transfusions containing the implicated RBC antigen

Cannot give blood again
if have received a blood transfusion

### 9. POST-TRANSFUSION PURPURA → very rare

→ occurs 5–12d post-transfusion

**Pathogenesis:**
- Host produces antibodies against donor platelets which destroy both the transfused platelets & patient's own platelets
- Results in severe, persistent thrombocytopenia

**Symptoms:** thrombocytopenia → purpura, bleeding

**Treatment:** IV immunoglobulins ± steroids

# IMMUNOLOGY AND ALLERGY

# 06

**Allergic disorders**....................82
**Autoimmune & immunodeficiency disorders**....83

## ABBREVIATIONS

**Ab** – Antibody
**ACEi** – Angiotensin-converting enzyme inhibitor
**ADCC** – Antibody-dependent cell-mediated cytotoxicity
**CVID** – Common variable immunodeficiency
**ELISA** – Enzyme-linked immunosorbent assay
**HBV** – Hepatitis B virus
**HCV** – Hepatitis C virus
**HIV** – Human immunodeficiency virus
**HSP** – Henoch–Schönlein purpura
**Ig** – Immunoglobulin
**MG** – Myasthenia gravis
**MGUS** – Monoclonal gammopathy of unknown significance
**SLE** – Systemic lupus erythematosus
**TXA** – Tranexamic acid

# Allergic disorders

**Common allergens:**
- nuts, shellfish, eggs, sesame seeds
- pollen, dust mites, animal fur
- insect stings

e.g. radioactive dyes

## Definitions

**Atopy:** ↑ IgE gives **genetic tendency** to develop allergies
**Allergy:** disease caused by **inappropriate immune response** to 'harmless' antigen
**Anaphylaxis:** potentially **life-threatening** type 1 allergic reaction → hypotension, ↑ HR, oedema, wheeze/stridor
**Anaphylactoid:** clinical presentation like anaphylaxis **but not IgE-mediated** → due to direct mast cell damage

## Classifying allergic disorders

| | | |
|---|---|---|
| **Type 1** | **IgE** crosslinking via allergen triggers **mast cell degranulation** | Asthma, food allergies |
| **Type 2** | **IgG** binds **cell-surface antigen** (ADCC) triggering **complement** reaction & cell lysis | MG, Goodpasture's |
| **Type 3** | **IgG** binds **free antigen → immune complexes** deposit in tissues | SLE, HSP |
| **Type 4** | **Cell-mediated** – T cells activate macrophages → inflammatory mediators & B cell activation (delayed reaction) | Contact dermatitis |

**Symptoms of allergies:**
- Rash
- Pruritus
- Swelling/angioedema
- Sneezing/rhinorrhoea
- Dyspnoea, wheeze, tachypnoea
- Nausea, vomiting, diarrhoea

**Long-term management of allergies:**
1. Avoid allergen
2. **Prescribe 2x EpiPens**
3. EpiPen training & education
4. Medi-Bracelet / alert device
5. Antihistamines if appropriate
6. **Good asthma control if have it**

## Investigations

→ *allergic disorders are usually a* ***clinical diagnosis***

1. **Detailed history** (most important)
    - Symptoms – timing, type, previous experience
    - Potential allergen identification – what? when? how much eaten?
    - DHx, PMHx, FHx – other atopies
2. **Skin prick test:** prick with antigen of allergen/s & measure size of rash
    - **Positive control:** histamine – may be negative if on antihistamines
    - **Negative control:** saline – only have reaction if **dermographism**

(skin reaction to trauma → **not allergy**)

3. **IgE testing:** if skin prick not conclusive
    - **Total IgE:** not specific to allergen
    - **Specific IgE**

Can measure baseline tryptase if suspect mast cell disorder (often associated with insect sting reactions)

4. **Challenge tests:** last resort / to prove negative → ONLY UNDER CLOSE MEDICAL SUPERVISION

*chronic urticaria = rarely allergic aetiology

**DDx of anaphylaxis:**
- ACEi angioedema (no urticaria)
- Hereditary angioedema
- Vasovagal episode
- Inducible laryngeal obstruction (ILO)
- Phaeochromocytoma
- Panic disorder
- Septic shock

**Management of allergic rhinitis**

| **Mild/intermittent** | **Severe/persistent** |
|---|---|
| - Antihistamines | - Antihistamines<br>- Intranasal steroid<br>- Chromone eyedrops |

## Specific reactions

| | **Urticaria** | | **Angioedema** |
|---|---|---|---|
| **Causes** | **Acute (<6w)**<br>- Idiopathic/spontaneous<br>- Concurrent infection<br>- **Allergic or anaphylactoid reaction** | **Chronic (>6w)***<br>- Idiopathic/ spontaneous<br>- Pressure, cold<br>- Dermographism | - **ACEis**<br>- Hereditary<br>- Viral infection<br>- Exercise, stress |
| **Management** | **Acute:**<br>- Consider oral corticosteroids<br>**Long-term:**<br>1. Avoid triggers<br>2. Antihistamines (if allergic cause) | | **Acute:**<br>1. **Stop ACEis** (if taking)<br>2. Oral corticosteroids<br>3. TXA (may help)<br>**Long-term:**<br>Avoid triggers ± antihistamines |

# Autoimmune & immunodeficiency disorders

**SPUR infections suggest immunodeficiency**
(**S**evere, **P**ersistent, **U**nusual, **R**ecurrent)

## Investigations

### 1. HISTORY (suggestive of autoimmune disorder)

- **PMHx or FHx:** autoimmune disorders / immunodeficiency / multisystem disease
- **Symptoms:** constant fatigue, arthralgia, myalgia
- **DHx:** penicillamine
- Female

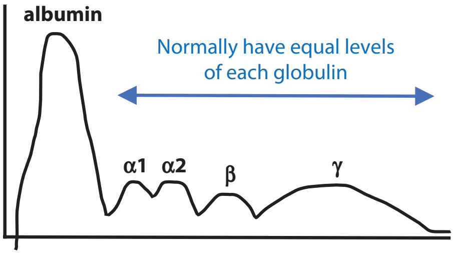


**Fig. 6.1** Serum electrophoresis.

### 2. BLOOD TESTS

| Autoimmune disease | Immunodeficiency |
|---|---|
| • **Urinalysis & BP**<br>• **FBC:** anaemia, leukopenia, thrombocytopenia<br>• **CRP/ESR** (ESR may be raised but not CRP)<br>• **U&Es, LFTs**<br>• **Coagulation screen**<br>• **Complement & Ig levels:** C3/C4 (↓ in SLE, immune-complex diseases & some infections)<br>• **Autoantibodies** (tested for with ELISA/immunofluorescence)<br>• **Blood-borne virus screen:** HIV, HBV & HCV serology<br>**Possible further Ix:** assess organ involvement<br>• Organ biopsy<br>• CXR / joint X-ray<br>• ECG | • **FBC:** lymphocyte subsets (T cells, B cells, NK cells) → recognise immunodeficiencies<br>• **Complement & Ig levels:** C3/C4 (↓ in SLE, immune-complex diseases & some infections)<br>• **Antibody levels:** IgG/A/M (↓ in myeloma & Ig deficiencies)<br>• **Functional antibody testing:** shows how well Igs function (measure Ig levels pre- & post-vaccination)<br>• **Serum protein electrophoresis:** (abnormal levels in myeloma & Ig disorders)<br>• **Urine protein electrophoresis:** Bence Jones protein<br>• **Blood-borne virus screen:** HIV, HBV & HCV serology |

**Examples of Ig disorders:**
- CVID
- Hyper IgM
- Hyper IgE
- MGUS
- Hypogammaglobulinaemia

*Cryoglobulinaemia = disorder where abnormal Igs become insoluble at cold temperatures*

## Summary of autoantibodies in different autoimmune disorders

| Condition | Autoantibodies |
|---|---|
| **Thyroid** | **Graves':** TSI (thyroid-stimulating immunoglobulin)<br>**Hashimoto's:** anti-TPO, anti-thyroglobulin |
| **Addison's disease** | Anti-21 hydroxylase |
| **Pernicious anaemia** | Anti-intrinsic factor |
| **Type 1 diabetes mellitus** | Insulin, GAD (glutamic acid decarboxylase) & islet cell autoantibodies (associated with HLA-DR3/4 alleles) |
| **Coeliac** | Anti-transglutaminase |
| **Goodpasture's syndrome** | Anti-type IV collagen (anti-basement membrane) |
| **Autoimmune hepatitis** | ANA, anti-SM, anti-LKM |
| **Primary biliary cholangitis** | Anti-mitochondrial |
| **Primary sclerosing cholangitis** | ANCA |
| **Rheumatoid arthritis** | RF, anti-CCP, anti-IgG |
| **Systemic lupus erythematosus** | ANA, RNP, dsDNA, anti-SM, anti-Ro, anti-La |
| **Anti-phospholipid syndrome** | Anti-lupus anticoagulant, anti-cardiolipin |
| **Sjögren's syndrome** | Anti-Ro (SSA), anti-La (SSB) |
| **Scleroderma** | Anti-centromere |
| **Pemphigoid** | Anti-desmoglein |

# NEUROLOGY

# 07

NERVOUS SYSTEM STRUCTURE & FUNCTION

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## ABBREVIATIONS

**#** – Fracture
**Ab** – Antibody
**ACA** – Anterior cerebral artery
**ACh** – Acetylcholine
**AED** – Anti-epileptic drug
**APS** – Antiphospholipid syndrome
**BBB** – Blood–brain barrier
**BZD** – Benzodiazepine
**CBT** – Cognitive behavioural therapy
**CIDP** – Chronic inflammatory demyelinating polyradiculopathy
**CJD** – Creutzfeldt–Jakob disease
**CK** – Creatine kinase
**CLD** – Chronic liver disease
**CMT** – Charcot–Marie–Tooth
**CMV** – Cytomegalovirus
**CN** – Cranial nerve
**CNS** – Central nervous system
**CSF** – Cerebral spinal fluid
**CTD** – Connective tissue disease
**CVST** – Cortical venous sinus thrombosis
**DCML** – Dorsal column medial lemniscus
**DIC** – Disseminated intravascular coagulation
**EBV** – Epstein–Barr virus
**EEG** – Electroencephalography
**EID** – Electronic implantable device
**EMG** – Electromyography
**GBS** – Guillain–Barré syndrome
**GCA** – Giant cell arteritis
**GCS** – Glasgow Coma Score/Scale
**HNPP** – Hereditary neuropathy with pressure palsies
**HSV** – Herpes simplex virus
**IC** – Intracranial
**ICA** – Internal carotid artery
**ICP** – Intracranial pressure
**ILAE** – International League Against Epilepsy
**IVH** – Intraventricular haemorrhage
**LMN** – Lower motor neurone
**LOC** – Loss of consciousness
**LP** – Lumbar puncture
**LSBP** – Lying & standing blood pressure
**MCA** – Middle cerebral artery
**MG** – Myasthenia gravis
**MND** – Motor neurone disease
**MS** – Multiple sclerosis
**N / n.** – Nerve
**NCS** – Nerve conduction study
**NMJ** – Neuromuscular junction
**OCP** – Oral contraceptive pill
**PCA** – Posterior cerebral artery
**PCR** – Polymerase chain reaction
**PNS** – Peripheral nervous system
**SAH** – Subarachnoid haemorrhage
**SAN** – Spinal accessory nerve
**SLE** – Systemic lupus erythematosus
**SOL** – Space-occupying lesion
**SSRI** – Selective serotonin reuptake inhibitor
**TCA** – Tricyclic antidepressant
**TIA** – Transient ischaemic attack
**UMN** – Upper motor neurone
**VDRL** – Venereal Disease Research Laboratory
**VF** – Visual field

# Areas of the brain

**Language centres:**

→ usually in **dominant hemisphere**

**1. Broca's area:** expression

→ **Area 44 & 45** (inferior frontal gyrus)

→ Broca's aphasia = **expressive dysphasia** (non-fluent speech)

**2. Wernicke's area:** reception

→ **Area 22** (superior temporal gyrus)

→ Wernicke's aphasia = **receptive dysphasia** (impaired language comprehension)

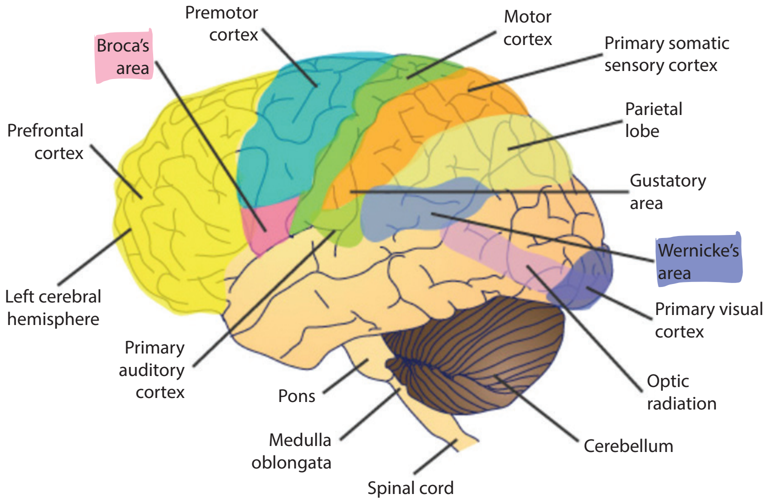

Premotor cortex
Broca's area
Motor cortex
Primary somatic sensory cortex
Prefrontal cortex
Parietal lobe
Gustatory area
Wernicke's area
Left cerebral hemisphere
Primary visual cortex
Primary auditory cortex
Pons
Optic radiation
Medulla oblongata
Cerebellum
Spinal cord

**Fig. 7.1** Surface anatomy of the brain.

## The cerebellum

→ ***function:*** *balance & coordination of* ipsilateral *muscles*

**Symptoms of cerebellar dysfunction:**

- Ataxia → broad-based gait (worse when heel-to-toe walking)
- Intention tremor → absent at rest
- Upward drift → with arms straight out in front, eyes closed
- Positive rebound phenomenon
- Dysmetria → cannot estimate distance (finger-to-nose test)
- Dysarthria → **slow, slurred 'staccato' speech** (say 'British Constitution')
- Dysdiadochokinesia → clumsy alternating rapid movements
- Nystagmus

**Causes of cerebellar damage:** 'PASTRIES'

**P** – Posterior fossa tumour
**A** – Alcohol
**S** – Multiple sclerosis
**T** – Trauma
**R** – Rare causes (neurodegenerative, paraneoplastic)
**I** – Inherited (e.g. Friedreich's ataxia)
**E** – Epilepsy treatments
**S** – Stroke

## The brainstem

→ ***function:*** *control of vital functions e.g. breathing*

**Midbrain: CN III & IV**
+ Edinger–Westphal nucleus
Vertical & horizontal eye movements

**Pons: CN V, VI, VII & VIII**
Horizontal eye movements & facial muscles

**Medulla: CN IX, X, XI & XII**
+ Vestibular nuclei (between pons & medulla)
(damage = vertigo)
+ Nucleus ambiguous
(damage = swallow issues)
+ Pyramidal decussation (spinothalamic)
(contralateral pain/temperature loss)
+ Respiratory & cardiac centres

**The cranial nerves**

| | | |
|---|---|---|
| **I** | Olfactory | Smell |
| **II** | Optic | Vision |
| **III** | Oculomotor | Eye movement |
| **IV** | Trochlear | Eye movement |
| **V** | Trigeminal | Face & anterior 2/3 tongue sensation<br>Chewing |
| **VI** | Abducens | Eye movement |
| **VII** | Facial | Taste (anterior 2/3 tongue)<br>Facial muscles<br>Lacrimal & salivary glands |
| **VIII** | Vestibulocochlear | Balance & hearing |
| **IX** | Glossopharyngeal | Taste (posterior 1/3 tongue)<br>Gag reflex |
| **X** | Vagus | Auricle, diaphragm, gut sensation<br>Uvula & swallowing (gag reflex)<br>Voice muscles<br>GIT autonomic functions |
| **XI** | SAN | Trapezius (turn neck & shrug) |
| **XII** | Hypoglossal | Tongue movement |

**KEY:**
**Sensory function**
**Motor function**
**Autonomic function**

# Vision

## Visual field defects

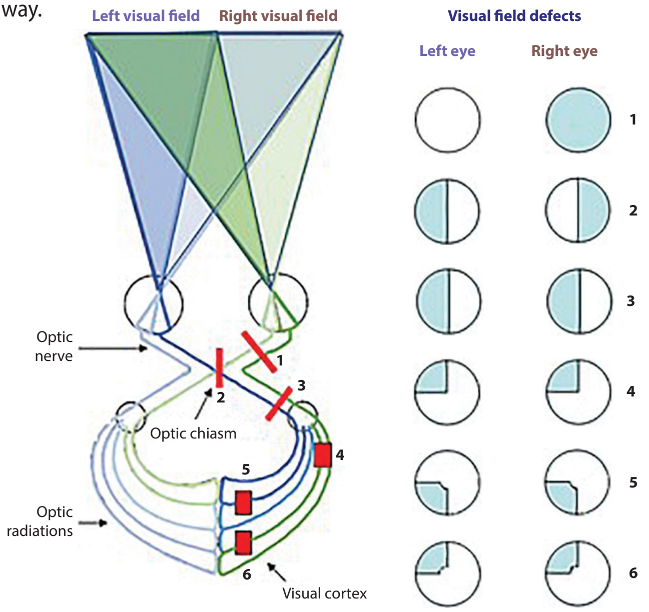
1. **Bitemporal hemianopia** = due to lesion in optic chiasm
2. Any **one-sided loss** (homonymous) = **retrochiasmal** & will be **contralateral** to lesion
3. **Quadrantanopias** = temporal or parietal lobe lesions → ask about other symptoms

**Extra-ocular muscles:**

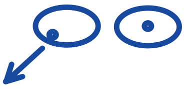
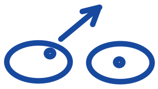
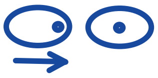
**SR** – superior rectus
**MR** – medial rectus
**IO** – inferior oblique
**IR** – inferior rectus
**LR** – lateral rectus
**SO** – superior oblique

1. **Monocular blindness**
2. **Bitemporal hemianopia**
   = **chiasm lesion**
   **Causes:**
   - Pituitary tumour
   - Rathke's pouch meningioma
   - Craniopharyngioma (kids)
3. **Homonymous hemianopia**
   = **optic tract, optic radiation, or occipital cortex lesion**
4. **Superior homonymous quadrantanopia**
   = **temporal lobe lesion**
   → **Inferior fibres** of optic radiation affected
5. **Inferior homonymous quadrantanopia**
   = **parietal lobe lesion**
   → **Superior fibres** of optic radiation affected
6. **Homonymous hemianopia + macular sparing**
   = **lesion of PCA but spared MCA**

**Fig. 7.2** Optic pathway.



## Eye muscle palsies

| | 3rd nerve palsy | 4th nerve palsy | 6th nerve palsy |
|---|---|---|---|
| **Symptoms/signs** | • Ptosis – levator palpebrae<br>• Down & out – SR, IR, MR, IO<br>• Efferent pupil defect – dilated | • Up & in – SO<br>• Compensatory head tilt away from affected eye | • Adducted – LR |
| **Causes** | **Pupil sparing:** stroke*, microvascular events, demyelination<br>**Non-pupil sparing:** tumour, orbital trauma, PCA aneurysm<br>*3rd nerve palsy due to stroke is usually accompanied by other symptoms | **Trauma** = most common<br>(+ other causes same as 3rd nerve palsy) | Trauma, tumour, demyelination, stroke*, microvascular events, idiopathic intracranial hypertension*<br>*6th nerve palsy can be a falsely localising sign of ↑ ICP |
| **Appearance** |  |  |  |

## Horner's syndrome

→ *arises due to disruption of sympathetic oculomotor supply to the eye*

**SYMPTOMS:**
- Unilateral **miosis** (pupil constriction)
- Partial **ptosis**
- **Anhidrosis**
- Apparent exophthalmos

**CAUSES:**
**Brainstem:** tumour, medullary stroke, MS, basal meningitis
**Intrathoracic lesion:** Pancoast tumour, cervical rib
**Neck lesion:** lymphadenopathy, trauma, thyroid surgery
**Intracranial:** ICA aneurysm, migraine

# Spinal cord tracts

**Corticobulbar tract innervates each cranial nerve bilaterally except for:**

1. **Facial nerve (CN VII):**
→ Forehead = bilateral representation
→ Branch to lower face = unilateral representation
→ UMN lesion spares frontalis

2. **Hypoglossal (CN XII):**
→ Each half of tongue supplied by contralateral corticobulbar tract
→ Lesion causes contralateral weakness, so tongue deviates **towards** weak side

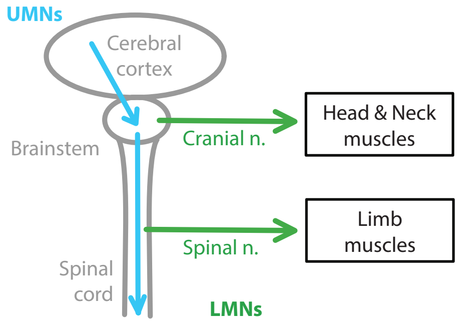


**Fig. 7.3** Pathway of UMNs & LMNs.

***Pyramidal weakness:**
**Upper limbs:** extensor muscles weaker than flexors
= **results in** flexed elbow & wrist
**Lower limbs:** flexor muscles weaker than extensors
= **results in** extended knee/ straight leg

**Bulbar palsy:** **LMN lesion** causing dysarthria = weakness of muscles supplied by CN IX, X, XII
→ **Nasal speech / dysarthria**
→ **Hyporeflexia:** jaw jerk & gag reflex absent
→ **Tongue:** weak & wasted & fasciculations

## The descending spinal tracts

**Pyramidal tracts** = voluntary control (cerebral cortex → brainstem & spinal cord)

| **Corticospinal:** voluntary movement of **contralateral body** | **Corticobulbar:** voluntary movement of **face & neck** |
|---|---|
| → Originate in **motor cortex**<br>→ **Decussate in medullary pyramids**<br>→ Synapse with **LMNs** in **spinal cord** | → Originate in **motor cortex**<br>→ Terminates in **brainstem**<br>→ Synapse with **cranial nerves** |

## The ascending tracts

| Dorsal column medial lemniscus (DCML) | Spinothalamic | Spinocerebellar |
|---|---|---|
| Fine touch, vibration, proprioception **(contralateral)** | Pain & temperature **(contralateral)** | Unconscious proprioception **(ipsilateral)** (awareness of position & movement of body parts in space without conscious thought) |

## Upper motor neurones vs. lower motor neurone lesions

**UMNs:** motor cortex → internal capsule → brainstem → spinal cord
**LMNs:** anterior horn cell → nerve root → peripheral nerve → NMJ → muscle

| | **Upper motor neurone lesion** (brain + spinal cord) | **Lower motor neurone lesion** (peripheral nerves) |
|---|---|---|
| **Signs** | • Hypertonia<br>• Hyper-reflexia<br>• **Pyramidal** weakness*<br>• Clonus<br>• +ve Babinski<br>• Spastic gait | • Hypotonia<br>• Hypo-reflexia<br>• **Proximal/distal** weakness<br>• Wasting<br>• Fasciculations |
| **Causes** | • Stroke / brain tumour<br>• Spinal tumour / injury<br>• Cerebral palsy / MS | • Peripheral neuropathy (DM, alcohol, drugs)<br>• Polio (anterior horn cells) / Guillain–Barré<br>• MND<br>• Myasthenia gravis |

## Facial weakness

| | **Upper motor neurone lesion** | **Lower motor neurone lesion** |
|---|---|---|
| **Signs** | • **Contralateral** weakness<br>• **Frontalis spared** | • **Ipsilateral** weakness<br>• **Whole face** |
| **Causes** | • Stroke / brain tumour<br>• Subdural haematoma<br>• MS | • Bell's palsy<br>• GBS (usually bilateral)<br>• Infection (HSV, CMV, EBV, Lyme disease)<br>• Trauma |

**Pseudobulbar palsy:** bilateral **UMN lesion** causing dysarthria = damage to medullary cranial nerves
→ **'Donald Duck' speech** (spastic dysarthria)
→ **Hyperreflexia:** jaw jerk & gag reflex increased
→ **Tongue:** weak & spastic

## Dermatomes

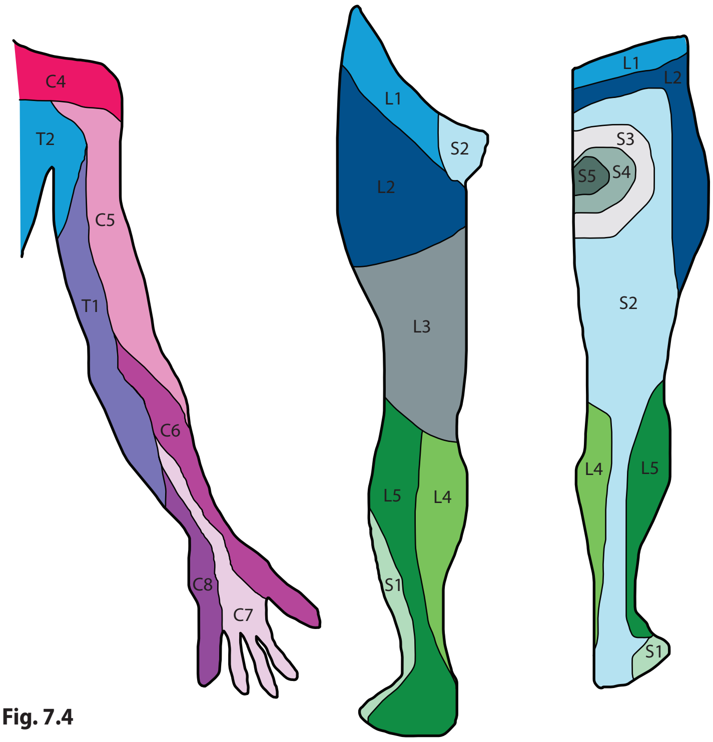


Fig. 7.4

## Myotomes

| Nerve root | Motor function |
|---|---|
| **C5** | Elbow flexion |
| **C6** | Wrist extension |
| **C7** | Wrist flexion, finger extension, elbow extension |
| **C8** | Finger flexion |
| **T1** | Finger abduction |
| **L1,2** | Hip flexion |
| **L3** | Knee extension |
| **L4** | Ankle dorsiflexion |
| **L5** | Knee flexion, big toe extension |
| **S1** | Knee flexion, ankle plantarflexion, big toe flexion |

| Deep tendon reflex | Nerve roots |
|---|---|
| **Biceps + brachioradialis** | C5, C6 |
| **Triceps** | C7, C8 |
| **Knee jerk** | L3, L4 |
| **Ankle jerk** | S1, S2 |

## Focal peripheral nerve lesions

| Lesion | Symptoms | Sensory loss | Motor loss | Causes |
|---|---|---|---|---|
| **Median nerve (C5–T1)**<br>Carpal tunnel | • Pain/tingling in hand<br>• **Thenar muscle wasting** | Dorsal & palmar aspect of lateral 3.5 digits | • Middle & index fingers **(L)**<br>• Thenar muscles **(OAF)**<br>**L**umbricals<br>**O**ppenens<br>**A**bductor pollicis brevis<br>**F**lexor opponens | • Repetitive motion<br>• DM/thyroid<br>• RA/OA<br>• Pregnancy |
| **Ulnar nerve (C8–T1)**<br>Cubital tunnel | • Pain/tingling in hand<br>• Weak grip<br>• **Ulnar claw (paradox)** | Dorsal & palmar aspect of medial 1.5 digits | **All small hand movement except LOAF (interossei/lumbricals)**<br>• Finger abduction & adduction<br>• Thumb adduction<br>• Little & ring finger flexion<br>• Wrist flexion | • Elbow trauma<br>• Idiopathic |
| **Radial nerve (C5–T1)** | • **Wrist drop** | Dorsal hand & 1st web space | **Brachioradialis:** Wrist extension<br>**Extensor digitorum:** Finger extension | • Humerus fracture |
| **Brachial plexus (C4–T1)** | • Erb's palsy (weakness, sensory loss & muscle atrophy) | Shoulder & arm | **Deltoid, biceps, brachialis, rotator cuff, serratus anterior** | • Idiopathic<br>• Trauma<br>• Damage from assisted delivery at birth<br>• Compression<br>• Inflammatory |
| **Axillary nerve (C5–C6)** | • Deltoid wasting | Regimental badge area | **Deltoid:** shoulder abduction | • Proximal humerus fracture |
| **Common peroneal nerve (L4–S2)** | • **Foot drop** | Lateral calf & dorsum of foot | **Tibialis anterior:** dorsiflexion & eversion | • Sporting injury (direct trauma near knee)<br>• Fibular head fracture<br>• Habitual leg crossing |

# Lumbar puncture

**Layers through which the needle passes:**
Skin → Subcutaneous tissue → Ligamentum flavum → Subarachnoid space

## Method

### 1. POSITION PATIENT

**→ Ideal position = lateral decubitus**

Patient lies on their side, curled forwards with their knees towards their chest to open the lumbar interspinous spaces. **This is the only way opening pressure can be measured.**

**→ If cannot lie on side / obese (or opening pressure not needed)**

Patient sits up, and curls forwards over a pillow.

**→ Cannot assess opening pressure if patient in sitting position**

### 2. FIND PUNCTURE SITE: L3/L4 or L4/L5 spaces

- **Identify L4 spine** – palpate tops of the iliac crests & L4 is halfway between (along line of the intercristal plane)
- LP needle **inserted obliquely above L4** – parallel to plane of the spine through the interspinous ligament

**Indications for LP:**

1. **Diagnosis of:**
    - **Infection** e.g. meningitis, encephalitis
    - **Neurodegeneration** e.g. CJD
    - **Inflammation** e.g. MS, GBS
    - **SAH** (if clinically suspected but no evidence on CT)
2. **Measurement of CSF pressure** (if no contraindication)
3. **Therapeutic removal of CSF**
4. **Intrathecal drug administration**

**Contraindications for LP:**

1. **Suspected raised ICP: will cause coning & death**
   e.g. posterior mass lesion, ventricular obstruction
   **Signs:** papilloedema, focal neuro deficit / seizures
2. **Increased bleeding risk:**
    - Anticoagulants – stop before LP
    - Thrombocytopenia (platelets <50)
    - Thrombophilias (INR >1.5)
3. **Intracranial abscess / severe septic features / meningococcal rash**

## Possible complications of LP

| Common | Rare |
|---|---|
| • Low pressure headache (30%)<br>→ worse when sitting up, better lying flat<br>→ typically resolves by 2w<br>→ treat with analgesics ± blood patch<br>• Backache (30%)<br>• Pain of anaesthetic & puncture<br>• Dry tap / unsuccessful procedure | • Infection<br>• Serious bleeding e.g. epidural haematoma<br>• Damage to spinal nerves / lasting neuro deficit<br>• Coning of the cerebellar tonsils / death<br>→ never do if suspect ↑ ICP due to SOL |

## Causes of raised opening pressure

→ Bacterial/viral infection
→ Cerebral haemorrhage
→ Idiopathic intracranial hypertension (pressure >25cm$H_2O$)
→ Venous sinus thrombosis

normal opening pressure <25cm$H_2O$

# Imaging with CT & MRI

## Common indications for brain imaging

- Trauma
- Tumour
- Stroke/TIA
- Neurodegeneration
- Inflammation
- Infection

→

**Patients who present with:**

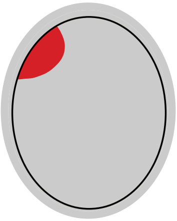
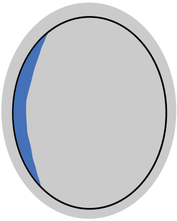
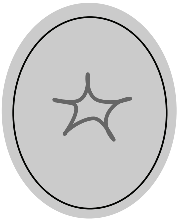
- Headache
- Vomiting
- Fever
- Seizure
- Low GCS / altered behaviour
- Focal neurology

**Head trauma plus:** 'panda eyes' / anticoagulated / vomiting / seizures

## CT versus MRI

| | CT scan | MRI scan |
|---|---|---|
| **Advantages** | • Quick, readily available<br>• Very sensitive for haemorrhage | • Non-ionising<br>• More detailed image / detects inflammation<br>• Safe in pregnancy after 1st trimester |
| **Disadvantages** | • Ionising radiation<br>• Not as detailed as MRI<br>• Not if pregnant | • Contraindications: metal implants / EIDs / claustrophobia<br>• Takes longer / less available<br>• More expensive |
| **Indications** | **In acute setting (quick):**<br>• assessment for early thrombolysis<br>• very sensitive for IC haemorrhage | • Small/subtle infarcts<br>• Posterior fossa assessment<br>• MS, epilepsy, tumours |

## CT imaging of intracranial haemorrhage

| **Epidural/extradural haematoma**<br>**Arterial blood** (middle meningeal artery) | **Subdural haematoma**<br>**Venous blood** (bridging veins) | **Subarachnoid haemorrhage** |
|---|---|---|
| Between skull & outer layer of dura | Between dura & arachnoid mater | Between arachnoid membrane & pia mater |
| **Sx:** lucid interval → unconscious | **Sx:** progressive headache & confusion | **Sx:** thunderclap headache |
|  **Fig. 7.5** |  **Fig. 7.6** |  **Fig. 7.7** |
| • **Lentiform / bi-convex shaped**<br>• Does not cross suture zones (confined)<br>• Midline shift | • **Crescent-shaped**<br>• Crosses suture lines<br>• Midline shift | • **Linear/star-shaped**<br>• Within sulci |

### Other neurological investigations

**Nerve conduction studies** (electromyography – EMG)

**Indications:**

- Polyneuropathy of unknown cause
- Mononeuritis multiplex
- Mononeuropathy that cannot be localised clinically
- Neuromuscular junction disorders / muscular disease
- Anterior horn cell disorders (e.g. MND)

**Electroencephalography (EEG)**

**Indications:**

- Seizures → determine type/diagnosis of epilepsy
- Assess for encephalitis

# Acute spinal problems

## Acute cauda equina syndrome

→ BILATERAL sciatica
→ Saddle anaesthesia (S2, S3, S4)
→ Bladder/bowel incontinence
= **emergency referral for MRI scan**

**Common cause:**
→ **central** lumbar disc herniation

## Cord compression

**Symptoms can develop over a few hours:**
→ Increasing thoracic back pain & local tenderness
→ Increasing leg weakness & incontinence

**Important cause: metastases**
→ from breast, prostate, lung, thyroid, renal, bowel

**Management:**
→ Urgent neurosurgical referral
→ MRI spine

## Acute spinal stroke

**Pathogenesis:** ischaemia or haemorrhage affecting vascular supply to spinal cord

**Symptoms:** (appear suddenly)
→ Severe neck/back pain
→ Leg weakness
→ Altered sensation
→ Bladder/bowel incontinence

**Management:**
→ MRI spine + stroke management

## Transverse myelitis

**Pathogenesis:** inflammation of spinal cord
**Causes:** idiopathic, autoimmune (MS), infection, systemic inflammatory (SLE)
**Investigation:** CSF analysis, antibody testing, spinal cord imaging

## Acute injury/compression of the spinal cord

1. Initial spinal shock = flaccid areflexia & paralysis, hypotension & bradycardia
2. Development of UMN signs: spasticity, hyperreflexia, increased tone etc.

# Neuromuscular emergencies

→ **Arise when weakness affects respiratory or bulbar muscle function**
→ **Can lead to type 2 respiratory failure**

## Underlying causes

1. **ACUTE:** Guillain–Barré, myasthenic crisis
   **Blood gas → high** $CO_2$ **+ low** pH & bicarbonate **= respiratory acidosis**
   (no time for compensation)
2. **CHRONIC:** MND, myotonic dystrophy, chronic myopathies
   **Blood gas → high** $CO_2$ **+ normal** pH & **high** bicarbonate
   (body compensates for respiratory acidosis over time)

## Clinical presentation of acute cases

- **Weak diaphragm** = breathless (especially when flat), accessory muscle use, paradoxical diaphragm movement
- **Weak intercostal/abdominal muscle**s = weak cough
- **Weak bulbar muscles** = cannot clear secretions, weak facial muscles
- **Signs of progressive hypercapnia & hypoxia** = tachycardia, sweating, flap → confusion

## Assessment

- **FORCED VITAL CAPACITY = best predictor of need for HDU**
  - In 70kg person, **<1.5L = serious concern**
  - **Drop of >20% from upright → supine** = diaphragm paralysis
- **General obs:** ↑ HR & RR
- **ABG:** respiratory acidosis
- **Neuro assessment:** weak limbs, face & swallow (cannot complete sentences / count from 1 to 20)

## Management

1. **Call for help (anaesthetist & ICU)**
2. **Secure airway:** sit up, $O_2$ if <94%, suction secretions, NBM
3. **IV access:** bloods & **blood gases**
4. **CXR:** for infection (common trigger)
5. **Neurology / critical care input for further management**

neuromuscular emergencies may be precipitated by infection

**ABG RESULTS**

| Acute T2RF | Chronic T2RF |
|---|---|
| → $O_2$ <8kPa<br>→ $CO_2$ >6kPa<br>→ pH <7.35<br>→ Bicarb = low/ normal | → $O_2$ <8kPa<br>→ $CO_2$ >6kPa<br>→ pH = **normal**<br>→ Bicarb = **>26mmol/L** |

**NB: Vital capacity normally decreases by 10% from upright → supine**

# Acute hydrocephalus

Accumulation of CSF within ventricles causing dilation & raised ICP

## Types

1. **Communicating (non-obstructive):** impaired CSF absorption → post-infection (TB), SAH, haemorrhagic stroke
2. **Non-communicating (obstructive):** blocked CSF flow between ventricles = ↓ drainage → tumours

## Symptoms

- Headache, N&V
- Blurred vision, papilloedema
- Ataxia (poor balance)
- Altered consciousness / sleepy
- Altered personality / irritable

→ bilateral UMN signs due to corticospinal tract disruption

## Investigations

- CT/MRI head
- Lumbar puncture

## Management

1. **Surgical:**
   - Shunt insertion
   - Endoscopic third ventriculostomy (ETV)
   - ± removal of obstruction if present
2. **Rehab:** physio, OT, psychology

### Causes of congenital hydrocephalus

- Genetic abnormality
- Developmental abnormality of brain/SC/spine
- Premature complications (IVH)
- Infection during pregnancy (e.g. rubella)

**Risk factors for acute hydrocephalus:**

- Congenital malformation of ventricular system
- Posterior fossa / brainstem tumours
- Post brain injury (SAH, meningitis, trauma)

### Normal pressure hydrocephalus:

= dilated ventricles with normal ICP (slow increase in CSF, allowing surrounding tissue to compensate so no ↑ ICP)

**Presentation:**

TRIAD: ataxia, incontinence, cognitive difficulties (DDx: Alzheimer's)

### Raised intracranial pressure (>25mmHg)

**Clinical presentation:**

1. **Headache of ↑ ICP**
   → new onset, progressive severity & frequency
   → associates with N&V
   → wakes patient early morning
   → worse with bending, coughing & exertion
2. **Vision changes:**
   → pupil constriction (later dilation)
   → reduced acuity & visual field loss
   → papilloedema
   → may have fixed, dilated pupil (3rd CN)
3. **Altered GCS:** irritability, drowsiness
4. **Cushing reflex** (due to **coning):** hypertension, bradycardia, Kussmaul breathing

**Investigations:**

- Hx & examination
- FBC, U&Es, LFT, glucose, clotting
- Blood culture
- CT head
- LP – **only if safe (opening pressure <25)**

**Causes:**

- Brain tumour
- Haemorrhage
- Hydrocephalus
- Status epilepticus
- Head injury
- Brain infection/ abscess
- Cerebral oedema

**Complications:**

- Brain herniation (coning)
- Ataxia
- CN palsies (esp. 3rd & 6th nerve)
- Irregular breathing / apnoea
- Hemiparesis
- Seizure/stroke/coma
- Infarction

**Management:** urgent neurosurgery (craniotomy / burr hole)

+ ABCDE ($O_2$, temp control)
+ seizure control
+ mannitol / hypertonic saline

# Cortical venous sinus thrombosis

A thrombus in the venous system draining the brain
→ results in venous congestion, hypoxia, and infarction

Rare: more common in people <50y

## Clinical presentation

### 1. RAISED ICP

- **Headache** – **usually thunderclap**, progressive & severe
- Seizures
- Papilloedema – visual blurring & VF loss
- CN VI palsy (NB: can be a falsely localising sign)

Suspect CVST if thunderclap headache + ↑ ICP, **without meningism**

### 2. FOCAL BRAIN INJURY:

Depends on area damaged (e.g. hemiparesis/aphasia)

**How to tell if it is a CVST or arterial cerebrovascular disease / stroke:**

**In CVST more likely to see:**

1. Seizures
2. Bilateral brain involvement
3. Reduced consciousness

**Differing appearance on CT/MRI:**

1. Do not correspond to arterial territories
2. Often have haemorrhage too

## Risk factors

1. **Prothrombotic conditions:** thrombophilia, protein C/S / antithrombin II deficiency, factor V Leiden, polycythaemia, APS
2. **Systemic inflammatory diseases:** IBD, sarcoidosis, SLE, Behçet's
3. **Cancer:** direct compression or hypercoagulability
4. **Infection:** meningitis / ENT infections
5. **Medications/drugs:** OCP, lithium, IVIg, IVDU
6. **Other:** pregnancy, head trauma, surgery, **dehydration** (↑Na, urea & Cr)

Consider CVST in young (female) patients with short Hx of headaches & seizures

## Investigations

- CT or MRI venogram – confirms diagnosis

## Management[1]

→ *alert seniors (contact ICU if necessary)*

1. **Anticoagulation:** LMWH + warfarin → despite concurrent haemorrhage as **thrombus is more dangerous**
2. **Lumbar puncture:** if signs of raised ICP → **safe if no shift effect despite ↑ ICP** (benefit of ↓ pressure on eyes)
3. **Thrombectomy** – may be offered
4. **Craniectomy** – only in a few cases where brain herniation & death is likely

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[1] Ferro *et al.* (2017) European Stroke Organization guideline for the diagnosis and treatment of cerebral venous thrombosis. *Eur J Neurol*, 24:1203.

# Stroke and cerebrovascular disease

**TIA:** acute focal neurological deficit of cerebrovascular cause **lasting <24h**
→ *usually resolves in minutes*

**STROKE: acute focal** neurological **deficit** of cerebrovascular cause **lasting >24h / results in death**

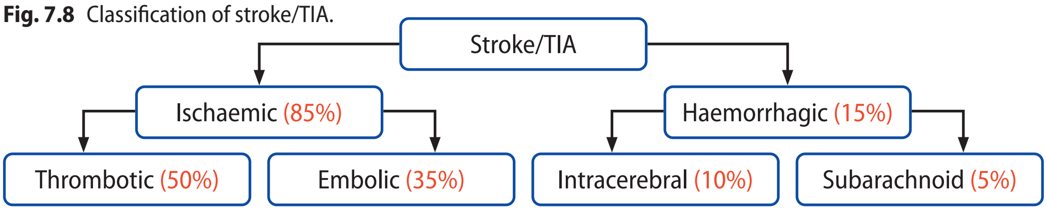


**Fig. 7.8** Classification of stroke/TIA.

**DDx of stroke/TIA:**
- Seizure (Todd's paresis)
- Migraine
- Functional neurological syndrome
- Transient global amnesia
- Metabolic disturbances
- Space-occupying lesion

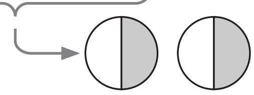
residual paresis post-seizure

***Highlights that symptoms of LOC &/or seizure make diagnosis of stroke very unlikely***

## SYMPTOMS OF ISCHAEMIC STROKE / TIA:

- **Motor:** unilateral weakness, slurred speech
- **Sensation:** numbness
- **Coordination:** impaired
- **Higher cognitive dysfunction:** dysphasia, agnosia, apraxia, inattention, memory loss
- **Vision:** amaurosis fugax, homonymous hemianopia

*painless, **transient loss of vision** in one eye → often described as a 'curtain descending'*



## SCREENING TOOLS:

**Outside of hospital:** FAST ('Face, Arm, Speech, Time')
**In A&E:** ROSIER ('Recognition Of Stroke In the Emergency Room')

**Other useful investigations:**
- ECG (consider AF as cause)
- Bloods: lipids, glucose, TFTs, clotting, FBC

## Transient ischaemic attack[2]

### IMMEDIATE MANAGEMENT OF SUSPECTED TIA:

1. Exclude hypoglycaemia as a cause
2. **300mg aspirin** (continue for 2w)
3. Referral to specialist **TIA clinic** to be seen **within 24h**

→ *CT not indicated unless suspicious of an alternative diagnosis*

| ICA stenosis results: | | |
|---|---|---|
| <50% | MILD | No intervention |
| 50–70% | MOD. | Consider carotid endarterectomy |
| >70% | SEVERE | Carotid endarterectomy |

### ONCE TIA CONFIRMED IN SPECIALIST CLINIC:

1. **Secondary prevention:** long-term clopidogrel (75mg) and atorvastatin (80mg)
2. **Control risk factors:** BP, cholesterol, blood glucose
3. **Consider carotid endarterectomy:** dependent on degree of internal carotid artery (ICA) stenosis

## Acute ischaemic stroke[2]

### IMMEDIATE MANAGEMENT OF ACUTE STROKE:

1. Exclude hypoglycaemia as a cause
2. Admission to **specialist stroke unit**
3. **CT HEAD** as soon as possible → confirm Dx & exclude haemorrhage
4. **300mg aspirin** once haemorrhage excluded (continue for 2w)
   - **Thrombolysis → if within 4.5h of onset** (NEED CLEAR ONSET TIME)
     - alteplase (tPA: tissue plasminogen activator)
   - **Thrombectomy → within 6h**
     - if LARGE CLOT in PROXIMAL anterior or posterior circulation
     - AND salvageable brain tissue seen on CT angiogram / MRI

**Contraindications of thrombolysis**

**Absolute:**
- Sx suggesting SAH
- Hx of IC haemorrhage/neoplasm
- GI bleed in past 3w
- Bleeding/clotting disorder
- BP >180/110
- Anticoagulated

**Relative:**
- Current cancer
- Age >75y
- Major surgery in past 2w
- Stroke **or** head trauma in last 3m

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[2] NICE (2019, updated 2022) *Stroke and transient ischaemic attack in over 16s: diagnosis and initial management* [NG128]

## WARD-BASED MANAGEMENT: ACUTE STROKE UNIT

1. **Ongoing monitoring:** *any deterioration in first 48h = REPEAT CT HEAD*
   → $O_2$, glucose, BP, pulse
   → Temp: ↑ of 1°C doubles ongoing brain damage
   → Skin assessment – for pressure sores
   → Swallow assessment – initial swallow assessment ASAP
2. **DVT prophylaxis:** IPC stockings (LMWH contraindicated as risk of bleed outweighs VTE risk reduction)
3. **SALT assessment** → *within 24h* (consider NG tube if unsafe swallow)
4. **Physiotherapy** → *within 24h* (relieve spasticity & prevent contractures)
5. **Occupational therapy** → *within 72h*

## SECONDARY PREVENTION

1. **Surgery:** carotid endarterectomy considered if ICA stenosis >50%
2. **Antiplatelet therapy:** CLOPIDOGREL (75mg PO OD long-term)
3. **Statins:** atorvastatin 80mg PO long-term (in all ischaemic stroke patients)
4. **ADDRESS RISK FACTORS**
   - BP – antihypertensives
   - AF – DOAC
   - Smoking, alcohol, diet, exercise, DM

## BAMFORD CLASSIFICATION OF STROKE[3]:

| | | |
|---|---|---|
| **TACS**<br>(20%) | **60% 1y mortality**<br>85% 1y morbidity | **All 3 of:**<br>• higher dysfunction (dysphasia, ↓ consciousness, inattention, neglect)<br>• homonymous hemianopia<br>• contralateral hemiparesis or hemisensory loss (≥2 of face, arm, leg) |
| **PACS**<br>(35%) | **15% 1y mortality**<br>45% 1y morbidity | **2 out of 3 of TACS criteria**<br>OR isolated higher dysfunction OR monoparesis |
| **LACS**<br>(20%) | **10% 1y mortality**<br>40% 1y morbidity | **1 of:**<br>• pure unilateral hemiparesis OR hemisensory loss<br>• pure unilateral hemi-sensorimotor loss<br>• ataxic hemiparesis (ipsilateral hemiparesis + cerebellar ataxia) |
| **POCS**<br>(25%) | **30% 1y mortality**<br>50% 1y morbidity | **1 of:**<br>• cranial nerve palsy PLUS contralateral motor/sensory loss<br>• cerebellar dysfunction (nystagmus, uncoordinated)<br>• bilateral motor or sensory deficit<br>• isolated homonymous hemianopia |

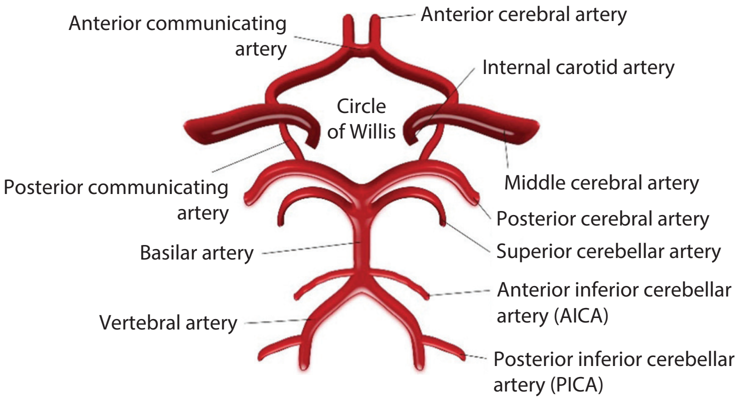
Anterior communicating artery
Anterior cerebral artery
Internal carotid artery
Circle of Willis
Posterior communicating artery
Middle cerebral artery
Posterior cerebral artery
Superior cerebellar artery
Basilar artery
Anterior inferior cerebellar artery (AICA)
Vertebral artery
Posterior inferior cerebellar artery (PICA)

**Fig. 7.9** Vascular supply of the brain.

**Follow current DVLA guidelines**

- **Cannot drive for minimum 4w**
- If ongoing deficit after 4w, must then inform DVLA

**Risk factors for ischaemic stroke/TIA:**

*Modifiable:*

- Hypertension
- AF
- DM
- Smoking
- Hyperlipidaemia
- BMI/obese
- Lack of exercise

*Non-modifiable:*

- Age
- Male
- FHx
- Previous stroke/ TIA

**Vessels affected in different strokes**

**TACS (total anterior circulation stroke):** MCA + ACA
**PACS (partial anterior circulation stroke):** small branches of MCA + ACA
**LACS (lacunar stroke):** small branches of MCA, basilar, vertebral
**POCS (posterior circulation stroke):** vertebral-basilar

MCA, middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery.

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[3] Bamford JM (2000) The role of the clinical examination in the subclassification of stroke. *Cerebrovasc Dis*, 10(suppl 4): 2–4

**Risk factors:**
- **Elderly**
- **Hypertension**
- Cerebral amyloid angiopathy
- AVMs
- Cerebral aneurysm
- Anticoagulated

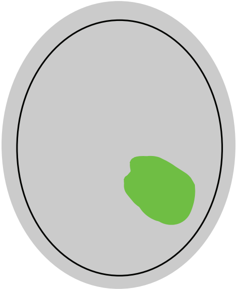

**Fig. 7.10** Diagrammatic representation of CT showing well-circumscribed intracerebral haemorrhage.

## Haemorrhagic stroke

### INTRACEREBRAL HAEMORRHAGE

1. **LOBAR:** in cerebral cortex → usually elderly (with cerebral amyloid angiopathy)
2. **DEEP:** in basal ganglia / brainstem / cerebellum → usually due to hypertension

### Symptoms

As for ischaemic stroke but the early presentation of the following is more likely:

- Severe headache
- Nausea, vomiting
- Seizures
- Decreased GCS
- Extremely high BP

### Investigations

**Consider underlying cause → tumour / vascular malformation / venous infarct**

1. CT → hyperdense collection within lobes
2. CT angiography → can show underlying vessel abnormalities
3. MRI → more reliably shows source of bleed

Surgery rarely considered

### Management[2]

1. **STOP ANTICOAGULANTS/ANTIPLATELETS** & reverse if needed
   - vitamin K for warfarin / protamine sulfate for heparins
2. **AGGRESSIVE BP MANAGEMENT:** **aim for SBP of ≤140**

   → unless there is an underlying structural cause (tumour, AVM, aneurysm)
3. **Discuss with neurosurgeons** → only operate if:
   - Midline shift (haemorrhage is space-occupying)
   - **AND** clinical signs of drowsiness / ↓ GCS

# CNS infection

## Meningitis

→ *inflammation of brain meninges*

**SYMPTOMS:**

- **Meningism:** headache, stiff neck, fever, photophobia ± vomiting, malaise, rigors
- **Altered mental state:** confusion/drowsiness/seizures
- **Focal neurological deficit**
- **Meningococcal rash** if *N. meningitidis*

**Kernig's sign:** pain with knee extension
**Brudzinski's sign:** hips flex if neck is flexed

**INVESTIGATIONS:**

1. **Bloods:** FBC, U&Es, LFT, CRP, clotting, glucose, lactate
2. **Blood cultures:** ideally before starting ABX **but do not delay Tx**
3. **Serum PCR:** meningococcal/pneumococcal PCR
4. **Throat swab / stool sample** → Consider HIV test
5. **Imaging:** CT/MRI → do not delay LP for these unless signs of ↑ ICP
6. **Lumbar puncture: within 1h unless contraindicated**
   → Opening pressure, WCC, protein, glucose, MCS, PCR
   → **Must measure serum glucose simultaneously to compare** (low CSF glucose is <50% of serum glucose)

**Bacterial causes**

- ***N. meningitidis* = most common**
- ***Strep. pneumoniae***
- *Haemophilus influenzae*
- *Listeria monocytogenes* = elderly/immunocompromised
- *E. coli* / Group B strep = neonates
- *Mycobacterium tuberculosis* = immunocompromised, malnourished, recent contacts

**Viral causes**

- Enteroviruses (Coxsackie)
- Herpes simplex / varicella zoster
- Epstein–Barr
- Mumps

**Fungal causes**

e.g. Aspergillus (immunocompromised)

**CSF FINDINGS:**

| | Bacterial | Viral | Fungal | TB | Encephalitis |
|---|---|---|---|---|---|
| **Opening pressure** | ↑ | Normal | Varies | Varies | ↑ |
| **WCC** | ↑↑ | ↑ | ↑ | ↑ | ↑ |
| **Cell type** | Polymorphs | Lymphocytes | Lymphocytes | Lymphocytes | Lymphocytes |
| **Protein** | ↑↑ | ↑ | ↑ | ↑ | ↑ |
| **CSF glucose** | ↓ | Normal | ↓ | ↓ | Normal |

**NB:** need to do additional Ix if suspect TB → Ziehl–Neelsen stain

**MANAGEMENT OF BACTERIAL MENINGITIS[4]:**

**start empirical ABX (do not wait for LP results)**

1. **IV ABX:** check local guidelines e.g. ceftriaxone ± amoxicillin for listeria cover →
2. **Steroids:** e.g. IV dexamethasone (reduces meningeal inflammation)
3. **Notify Public Health** (+ one-off ciprofloxacin dose for close contacts)

+ IV aciclovir if suspect concurrent encephalitis (altered mental state)

**COMPLICATIONS OF BACTERIAL MENINGITIS:**

| Acute | Longer-term |
|---|---|
| • Sepsis & DIC<br>• Hydrocephalus<br>• Venous sinus thrombosis<br>• Adrenal haemorrhage & dysfunction<br>(Waterhouse–Friderichsen syndrome) | • Abscess<br>• Seizures<br>• Ataxia<br>• Cognitive difficulties<br>• Cranial nerve palsy<br>(sensorineural hearing loss, visual impairment) |

**Cerebral abscess:**

**Cause:** *Staph. aureus* = common

**Symptoms:** mimics encephalitis
→ often preceding illness

**Diagnosis:** CT/MRI

**Management:** long course ABX
± neurosurgery

## Viral encephalitis

→ *inflammation of brain parenchyma*

| Features | Meningitis | Encephalitis |
|---|---|---|
| Fever, headache, N&V | Yes | Yes |
| Photophobia, neck stiffness | **Yes** | No |
| Seizures / altered mental state* | Late sign | **Yes** |
| CN palsies, paralysis, neuro deficit | Yes | Yes |

*Determine baseline mental state

Viral encephalitis usually caused by HSV-1

**INVESTIGATIONS:** same as meningitis
→ **Opening pressure & viral PCR from LP = key**
→ **MRI** shows areas of oedema & r/o abscess

**MANAGEMENT: often a lot more unwell** ↗ **Mortality = 70% if untreated**
→ **ABCDE** (manage if unconscious / treat seizures with BZDs)
→ **IV aciclovir** for 2w

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[4] McGill *et al.* (2016) The UK joint specialist societies guideline on the diagnosis and management of acute meningitis and meningococcal sepsis in immunocompetent adults. *J Infect*, 72:405.

# Brain abscess

A contained collection of organisms (bacteria, fungi or parasite) within the brain

## Aetiology

1. **Contiguous focus of infection:** sinusitis, otitis media, dental infection
2. **Haematogenous spread from more distant infection focus:** endocarditis, lung abscess
3. **Direct infection:** post-neurosurgical procedure, trauma

## Clinical presentation

- Persistent, progressive headaches
- Fever – absence does not exclude abscess
- Altered mental state
- Seizures
- Neurological deficit (signs of ↑ ICP)
- ↑ Head circumference – important in infants
- Bulging fontanelles – important in infants

Headache, cranial nerve palsies, papilloedema

## Investigation[5]

1. **Careful history:** symptom onset more acute than neoplasm
2. **Examination:**
   - Source of infection (ears, nose, mouth, IVDU, endocarditis)
   - Signs of ↑ ICP (cranial nerve palsy, papilloedema, anisocoria)
3. **Bloods:** ↑ WCC, ↑ CRP
4. **Blood culture** – may be positive if bacteraemia as source of infection
5. **CT head** – often first line
6. **MRI with contrast** – more detailed and sensitive than CT head

### OTHER INVESTIGATIONS TO CONSIDER:

- **Lumbar puncture** – if highly suspicious of toxoplasma infection or meningitis → perform with caution if ↑ ICP
- **CT CAP** – if suspecting malignancy

## Management[5,6]

1. **Empiric IV ABX** (in keeping with local guidelines)
   → If later confirmed fungal or parasitic source, switch to antifungal or antiparasitic treatment
2. **Anticonvulsant**
3. **Corticosteroid** e.g. dexamethasone – if severe cerebral oedema
4. **Surgical: contact neurosurgeon if diagnosis suspected after initial imaging**
   - Urgent decompression – if neurological decompensation
   - Excision – if >2.5cm or smaller and resistant to medical therapy

**Follow-up:** contrast CT head at increasing intervals

**Differentials:**
- CNS neoplasm
- Metastatic lesion
- Radiation necrosis post-op
- Encephalitis/meningitis
- Multiple sclerosis
- Ischaemic stroke

**Risk factors for brain abscess:**
- Immunocompromised/HIV
- IVDU/haemodialysis
- Diabetes mellitus
- Congenital heart disease
- Pulmonary AV malformations
- Recent neurosurgery
- Penetrating head trauma

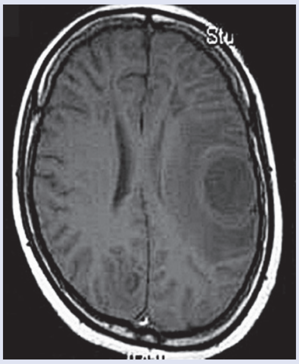

**Fig. 7.11** Brain abscess on MRI.

**Complications of brain abscess:**
- Seizures
- Hydrocephalus
- Ventriculitis
- Hyponatraemia
- Cognitive dysfunction
- Intraventricular rupture
- Brain herniation & death

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[5] Miranda *et al.* (2013) Brain abscess: current management. *J Neurosci Rural Pract*, 4: S67
[6] BMJ Best Practice (2021) *Brain Abscess*

# Neuro-oncology

## Primary brain tumours

| Tumour | Epidemiology | Prognosis |
|---|---|---|
| **Glioblastoma*** | Most common adult brain tumour | Poor: **malignant:** 2y average survival |
| **Meningioma*** | Most common brain tumour overall | Good: **benign:** surgery = curative |
| **Astrocytoma*** | Younger patients (≤20y) | Good: **benign:** surgery = curative<br>→ can become malignant |
| **Oligodendroglioma** | Older patients | OK: **malignant:** but chemo sensitive |
| **Medulloblastoma** | Children (occurs in posterior fossa) | OK: **malignant:** 70% 5y survival |

*most common adult brain tumours

### PRESENTATIONS OF BRAIN TUMOURS:

1. **Headache of ↑ ICP**
   → New onset, progressive severity & frequency
   → Associates with N&V
   → Wakes patient early morning
   → May have papilloedema
2. **Change in behaviour / new confusion**
3. **Progressive CNS signs** e.g. weakness, dysarthria, cranial nerve palsies (depends on location of mass)
   → key to differentiating from stroke is that they are **progressive**
4. **New seizures:** needs urgent neuro opinion
   → 6–10% of adult onset epilepsy is due to space-occupying lesion
   → Seizures particularly common in **temporal lobe lesions**

### MANAGEMENT: liaise with oncology

→ Consider oral dexamethasone if significant oedema surrounding lesions on CT

**Sources of secondary brain tumours:**

- Lung
- Renal
- Breast
- Melanoma
- Colon
- Thyroid

**But not prostate** (does not cross blood–brain barrier)

**Frontal lobe signs on examination:**

Return of primitive reflexes:

- Grasp reflex
- Pout reflex
- Palmomental reflex

**Important investigations:**

1. **History:** progressive symptoms
2. **Ophthalmoscopy:** ± papilloedema
3. **CT/MRI**
4. **Biopsy**

# Multiple sclerosis

**F>M = 3:1**
**Peak onset** = 20–40y
Higher incidence in **northern hemisphere**

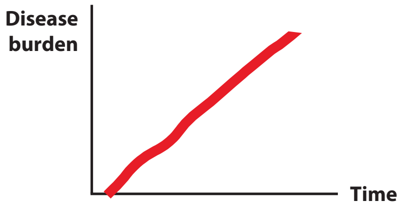
**Differentials of multiple sclerosis:**

1. **Other neuroinflammatory diseases:**
    - Vasculitis (SLE, Sjögren's, Behçet's)
    - Neurosarcoidosis
    - Neuromyelitis optica
    - Anti-MOG disease
2. **Infections:** Lyme disease, syphilis, HIV
3. **Cerebrovascular disease:** TIA/stroke
4. **MND**

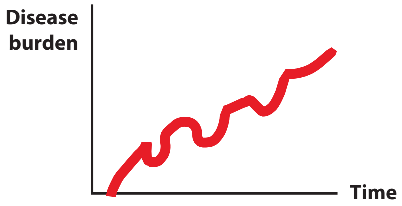
- Chronic, inflammatory, demyelinating disease of **CNS** (brain & spinal cord)
- Immune-mediated demyelination throughout the CNS
- Cause = unknown (thought to be multifactorial) → HLA-DR2 association

## Classification

1. **Primary progressive: 15%**
→ Slow and constant deterioration



2. **Relapsing–remitting: 85%**
→ Complete or incomplete recovery between episodes
→ 2/3 become secondary progressive



## Clinical presentation

| | |
|---|---|
| **Optic nerves** | • Optic neuritis (visual blurring / vision loss, pain on movement, ↓ colour differentiation) |
| **Brainstem** | • Diplopia / facial weakness or pain / internuclear ophthalmoplegia (INO) |
| **Spinal cord** | • Bladder/bowel/sexual dysfunction |
| **Dorsal columns** | • Tingling, paraesthesia & loss of proprioception ('useless hand syndrome') |
| **Corticospinal tracts** | • Paraparesis<br>• UMN deficit (i.e. increased tone + hyperreflexia) |
| **Cerebellar signs** | • Intention tremor, nystagmus, vertigo, dysarthria |
| **Long-term complications** | • **Excessive fatigue,** depression, **memory loss** |

## Investigations

1. **Neurological examination:** shows multifocal neurological deficits as per McDonald's criteria

**McDonald's criteria:**

a) **2 characteristic episodes** of neuro dysfunction **separated in time**
b) deficits represent lesions in **at least 2 different areas of CNS** e.g. hemisphere + optic nerve

2. **Bloods:**
    - FBC, U&Es, LFT, ESR, TFT, glucose, calcium, B12 → to r/o DDx
    - Autoimmune screen including anti-MOG Ab & aquaporin 4 Ab
    - Syphilis, Lyme disease & HIV serology
3. **MRI:** hyperintense lesions in periventricular white matter → key to Dx
4. **LP & CSF analysis:** if MRI inconclusive
    - Moderately increased or normal protein (still <1g/L)
    - Moderately raised or normal WBCs (still <50/mm$^3$)
    - Oligoclonal bands on electrophoresis → only present in CSF (not always present in MS)
5. **Neurophysiology tests:** visual evoked potentials
    - Delay in conduction velocity in affected eye when presented with visual stimuli

## Investigating a relapse episode

**Relapse** = acute onset of a focal/multifocal demyelinating event lasting ≥24h

1. **History:** part of body not functioning as normal for >24h (e.g. visual disturbance, paresis)
2. **Neuro exam**
3. **MRI:** may show new lesion

## Acute event / relapse management

**Aim:** shorten duration & severity of attack

**Mx = High dose corticosteroids:** methylprednisolone PO or IV (plus bone protection & PPI cover)
↳ call neuro/specialist team for advice

## Long-term management[7]

1. **Disease-modifying Tx:** immunotherapies
   IFN-beta (immunomodulator), immunosuppressants (dimethyl fumarate), MAbs
2. **Symptomatic Mx:**
   - Fatigue (amantadine)
   - Neuropathic pain (TCAs)
   - Spasticity (baclofen, BZDs)
   - Depression (CBT, SSRI)
   - Bladder/bowel dysfunction (anticholinergics/laxatives)

## Prognosis

- Life expectancy 6–10y less than normal
- 50% need walking support in 15–20y

**Poor prognostic factors:**

- Early cerebellar signs
- Older age at onset
- High MRI lesion load at onset

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[7] NICE (2014, updated 2019) *Multiple sclerosis in adults* [CG186]

# Epilepsy

Epilepsy is defined by the ILAE as **≥2 unprovoked seizures, occurring ≥24h apart** OR **1 unprovoked seizure and evidence of high risk of seizure recurrence**[8]

## Classifying seizures

**Seizures** = sudden disturbance of neurological function due to bursts of **abnormal, uncontrolled electrical activity**, resulting in a change in behaviour

1. **Epileptic seizure:** seizure due to **excessive, hyper-synchronous neuronal discharge** in the cerebral cortex
2. **Non-epileptic attack:** seizure-like events not involving abnormal electrical activity in the brain

| Epileptic seizures | Non-epileptic attack |
|---|---|
| • **Idiopathic** (70–80%) – presumed genetic<br>• **Secondary**<br>► Cerebral dysgenesis<br>► Cerebral vascular occlusion<br>► Cerebral damage e.g. hypoxia/infection<br>► Cerebral tumour<br>• **Neurodegenerative disorders**<br>• **Neurocutaneous syndromes** e.g. neurofibromatosis, tuberous sclerosis | • **Psychogenic/dissociative**<br>• **Syncope**<br>► Vasovagal<br>► Orthostatic<br>► Cardiac arrhythmia |
| • **Metabolic:** ↓glucose/Ca/Mg/Na<br>• **Poisons/toxins**<br>• **Febrile seizures** (children) | • **Head trauma**<br>• **Meningitis/encephalitis** |

## Investigating seizures

1. **History = most important →** need eyewitness account
   - Frequency, triggers, length, symptoms
   - Birth history
   - Any impairments, educational/psychological/social impacts of seizures
   - **Video seizure if possible**
2. **Examination**
   - CNS & PNS examination
   - CVS & respiratory examination – **important to r/o cardiac syncope**
   - Skin markers for neurocutaneous syndromes
3. **EEG** – interictal EEG can be normal in patients with epilepsy
   - Can show neuronal hyperexcitability in epilepsy (sharp wave complexes/spikes **and** slow wave abnormality)
   - If normal consider sleep-deprived / 24h EEG **if uncertain of diagnosis**
4. **Imaging: usually normal in epilepsy** (can show **cause** but not diagnostic)
   - MRI/CT – **gold standard investigation** to r/o tumour or CVD
   - PET/SPECT – detect areas of hypo-/hypermetabolism for epilepsy surgery assessment
5. **Tests to rule out other causes**
   - ECG – cardiac causes
   - Bloods – metabolic disturbances (**glucose**)

**Collateral history is the key** in reviewing patients with suspected seizures. **Always consider the possibility of non-epileptic attacks** in patients who do not respond to epilepsy medications. **Non-epileptic attacks can co-exist in patients with epilepsy.**

**Psychogenic/dissociative seizure:**
→ real events!
**Triggers:** previous trauma, flashing lights, emotions (stress, anxiety, depression)

**Vasovagal syncope:**
LOC due to abnormal autonomic response & temporary ↓blood supply to brain

**Features:** nausea, sweating, pallor, transient LOC ± clonic movements

**Triggers:** hot room, standing for long periods, fear

**Ix for syncope:**
1. LSBP
2. ECG
3. Tilt table (+ve >1 time)

**Cardiac red flags**
- Symptoms on exertion
- Palpitations
- Vascular RFs
- FHx of cardiac disease

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[8] Fisher RS *et al.* (2014) ILAE Official Report: a practical clinical definition of epilepsy. *Epilepsia*, 55(4): 475–82.

## Types of epileptic seizure[9]

**1. GENERALISED:** discharge from **both** hemispheres
→ No warning
→ Always have LOC

**2. FOCAL:** discharge from **one** part of **one** hemisphere
→ May be preceding aura
→ May or may not have LOC (focal aware **or** impaired awareness)
→ May lead to generalised tonic–clonic seizure if LOC

| **Generalised onset** | | |
|---|---|---|
| **Motor onset** | **Tonic–clonic** | **Tonic phase:**<br>• Stiffening / ↑ tone (may fall to ground)<br>• Hold breath → cyanosis<br>**Clonic phase:** **seconds–minutes**<br>• Rhythmical jerking<br>• Irregular breathing, cyanosis, salivation<br>• Tongue-biting, incontinence<br>→ Post-ictal drowsiness/confusion<br>**Atonic phase:**<br>• Loss of muscle tone ± jerking → fall |
| | **Tonic** | |
| | **Clonic** | |
| | **Atonic** | |
| **Non-motor** | **Typical absence** | • 'Blanking-out'/staring<br>• Absence of motor symptoms<br>→ Brief onset & termination (last seconds) |
| | **Atypical absence** | • More gradual onset<br>• Longer lasting (up to few mins)<br>• May have post-ictal symptoms e.g. confusion |
| | **Myoclonic** | • Sudden, repetitive, jerky movements of upper limbs, head and body **(spill/drop things** → occasionally fall) |

| **Focal onset** | | |
|---|---|---|
| **Motor onset** | • **Automatisms** (lip smacking/chewing, hand movements)<br>• Spasms<br>• Tonic/clonic/atonic seizures<br>clonic movements spreading proximally = **Jacksonian March** & suggests onset in primary motor cortex | |
| **Non-motor onset** | • Sensory (e.g. smell, taste, touch, vision)<br>• Autonomic (e.g. nausea) | • Cognitive<br>• Behavioural arrest |

**Advice for relatives during a seizure:**

**Do:** place pillow under head, recovery position, move nearby objects, note time seizure began → **buccal midazolam / PR diazepam if >5min if available**

**Do not:** restrict/move them, give food/drink until recovered

## Investigations for epilepsy

*→ Hx & video most important*

- **EEG** indicated if epilepsy suspected / after 1st seizure → can help determine type & quantify risk of seizure recurrence
- **Brain scans usually normal** (unless a visible cause of focal seizures e.g. stroke)

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[9] ILAE (2017) *ILAE Classification of the Epilepsies*

## Management of epilepsy[10]

1. **Education & advice:** specialist epilepsy nurse
   - Avoid baths / open water swims, alcohol & sleep deprivation
   - **Driving**
   - Conception counselling
2. **Anti-epileptic drugs:** until seizure-free for 2y
   - Aim for **monotherapy** at **lowest effective dose**
   - Counsel on side-effects
3. **Rescue therapy (BZDs)** **– call 999 if need to use**
   - Given to terminate prolonged seizures (>5min)
   - **Buccal midazolam, PR diazepam, PO clobazam** (for focal seizures)
4. **Other options** if refractory epilepsy
   - Vagal nerve stimulation
   - Surgery e.g. lobectomy

| AED | Indication | Side-effects |
|---|---|---|
| **Sodium valproate** | Generalised (1st /2nd line)<br>**Avoid in women of child-bearing age** | • Weight gain<br>• Hair loss<br>• **Teratogen** |
| **Lamotrigine** | Generalised (1st /2nd line)<br>Focal (1st line) | • Rash (SJS)<br>• Insomnia<br>• Headaches |
| **Levetiracetam** | Focal<br>Generalised | • Irritability |
| **Carbamazepine** | Focal | • Rash<br>• Hyponatraemia<br>• Ataxia<br>• CYP140 inducer |
| **Ethosuximide** | Absence | • N&V |

A single seizure does not indicate starting AEDs (start if >2 seizures or 1 seizure with abnormal EEG/MRI brain)

- **Must inform DVLA**
- 6m driving ban after 1st seizure
- If >1 seizure **must be 12m seizure-free** (includes isolated auras)

**Status epilepticus** = seizure >5min or repeated seizures with no recovery of consciousness in between

**Mx:** IV BZD → IV AED → anaesthetic + ICU

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[10] NICE (2012, updated 2021) *Epilepsies* [CG137]

# Neuropathies

Pathological process affecting a peripheral nerve/nerves (LMN ONLY)

## Causes of peripheral neuropathy

| | |
|---|---|
| **Demyelination** | • Antibodies against myelin – RA, CTDs, myxoedema<br>• Inflammatory – Guillain–Barré, chronic inflammatory demyelination<br>• Inherited – Charcot–Marie–Tooth Type 1 |
| **Axon degeneration** (from distal → proximal) | • Metabolic – DM, B12/folate deficiency, CKD/CLD (urea/ammonia build-up)<br>• Toxins – alcohol, lead, radiation, drugs<br>• Antibodies against axons – RA, CTDs<br>• Inherited – Charcot–Marie–Tooth Type 2 |
| **Compression** | • Entrapment of nerve e.g. carpal tunnel |
| **Infarction** | • Of vasa nervorum e.g. DM, arteritis |
| **Infiltration** | • Granuloma, sarcoid, cancer |

**Most common causes:**

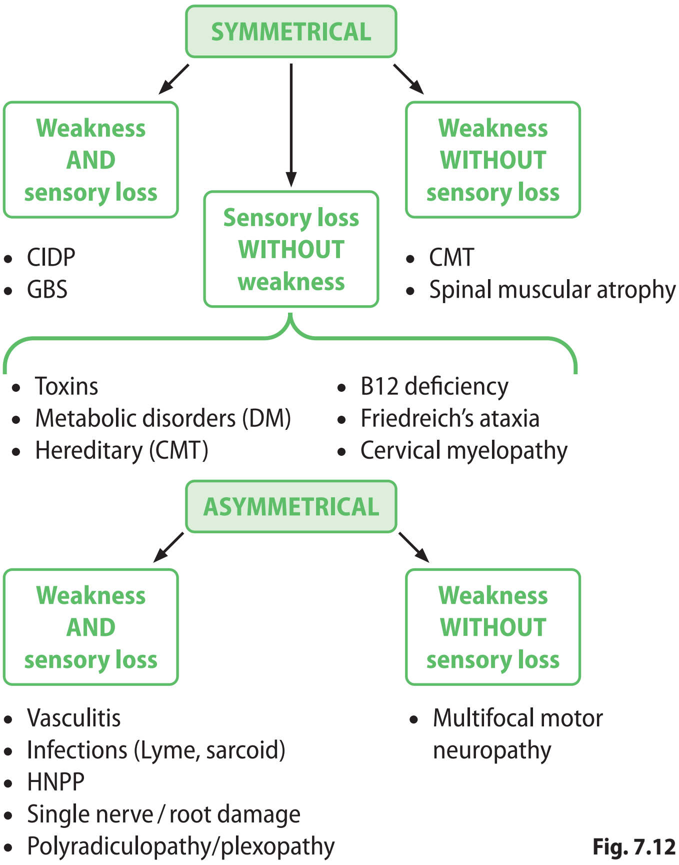
1. Diabetes
2. Vit B deficiency (affects DCML)
3. Drugs/toxins
4. Alcohol
5. Demyelination (CIDP/GBS)
6. Idiopathic (50% of cases)

## Investigations

- **Examination:** peripheral weakness, sensory loss & reflex loss **(LMN signs)**
- **Bloods:** FBC, ESR, U&Es, LFTs, glucose/HbA1c, B12/folate
- **Paraproteins & antibodies:** ANCA, VDRL test
- **Myeloma screen**
- **Neurophysiology testing** (EMG & NCS)
- **LP:** raised protein in GBS/CIDP
- **Peripheral nerve biopsy:** if diagnosis uncertain

*Neurophysiology testing: electromyography (EMG) & nerve conduction study (NCS)*

## Summary of patterns of neuropathy



**SYMMETRICAL**

**Weakness AND sensory loss**
- CIDP
- GBS

**Sensory loss WITHOUT weakness**
- Toxins
- Metabolic disorders (DM)
- Hereditary (CMT)
- B12 deficiency
- Friedreich's ataxia
- Cervical myelopathy

**Weakness WITHOUT sensory loss**
- CMT
- Spinal muscular atrophy

**ASYMMETRICAL**

**Weakness AND sensory loss**
- Vasculitis
- Infections (Lyme, sarcoid)
- HNPP
- Single nerve / root damage
- Polyradiculopathy/plexopathy

**Weakness WITHOUT sensory loss**
- Multifocal motor neuropathy

**Fig. 7.12**

## Guillain–Barré syndrome

*→ most common **acute** polyneuropathy*

**PATHOGENESIS:** viral infection triggers inflammatory response & **autoimmune demyelination**

**SYMPTOMS:** 1–3w after viral infection e.g. gastroenteritis / strep throat

- **Ascending paralysis** – starts in legs → trunk → arms
- Loss of tendon reflexes
- Facial weakness
- Bulbar weakness – dysarthria, dysphagia
- Respiratory muscle weakness – acute respiratory failure
- ± autonomic features (postural hypotension, arrhythmias)

A **motor dominant neuropathy** → sensory loss = uncommon

**INVESTIGATIONS:** **Dx usually on clinical Sx**

- **NCS:** slowed (demyelination)
- **LP:** raised protein (usually normal cell counts)

**MANAGEMENT[11]:**

- **Urgent** – rapidly progressing paralysis → ventilatory failure
- **High dose IV immunoglobulin** / plasma exchange
- May need ventilatory support (on HDU/ ICU)
- **Rehab:** physio, OT, SALT (consider keeping NBM)

**Prognosis:**

≈**60%** = full recovery in 6m
≈**30%** = remaining disability
**5–8%** mortality

**Complications:**

- Pneumonia
- SIADH
- Pseudo-obstruction

**Miller Fisher syndrome:** similar to GBS & can also present **after diarrhoeal illness**
→ **Classic triad of Sx:** ophthalmoplegia, areflexia, ataxia

## Differential diagnosis of neuropathy

### CHRONIC INFLAMMATORY DEMYELINATING POLYRADICULOPATHY

**Pathogenesis:** antibody-mediated demyelination

**Onset:** over months

**Symptoms:** progressive/relapsing **prox & distal weakness and sensory loss**

**Management:** immunosuppression

### MULTIFOCAL MOTOR NEUROPATHY

**Pathogenesis:** antibody-mediated **multifocal** demyelination e.g. vasculitis

**Onset:** over months

**Symptoms:** progressive **asymmetrical distal limb weakness** (starts hands/feet) = difficulty gripping, foot drop

### HEREDITARY SENSORIMOTOR NEUROPATHY (CHARCOT–MARIE–TOOTH)

**Pathogenesis:** genetic condition causing **axonal degeneration** (onset in childhood or 40s)

**Symptoms:**

- Progressive **symmetrical numbness, tingling & weakness** (starting in hands/feet & spreads proximally)
- Clawed toes, high arches, foot drop, muscle wasting
- Family history (not always)

**Investigations:**

- NCS = **slowed** due to demyelination ± axonal damage → rules out diabetes

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[11] BMJ Best Practice (2022) *Guillain–Barré Syndrome*

### DIABETIC NEUROPATHY

**Symptoms:** symmetrical distal sensory loss (can be painful/autonomic types)

### B12 DEFICIENCY

**Pathogenesis:** degeneration of dorsal & lateral white matter of spinal cord

**Symptoms:**

- Sensory ataxia & subacute combined degeneration of the spinal cord
- Progressive weakness + paraesthesia

## Trigeminal neuralgia

*→ facial pain experienced along the distribution of the trigeminal nerve*

### AETIOLOGY:

Idiopathic compression of trigeminal nerve (often by vascular loop of superior cerebellar artery) **or** demyelination

### CLINICAL PRESENTATION:

- **Facial pain** – sharp/stabbing episodes ± a constant background pain
- **No associated neurological deficit**

### INVESTIGATIONS: a clinical diagnosis

- CT/MRI

### MANAGEMENT[12]:

**1st line:** anticonvulsants (carbamazepine/oxcarbazepine)
**2nd line:** gabapentin, pregabalin, amitriptyline, nortriptyline, lamotrigine
**Medication-resistant:** microvascular decompression / ablative surgery

**Risk factors for trigeminal neuralgia:**

- Increased age
- Multiple sclerosis

**Differentials of trigeminal neuralgia:**

- Dental pain
- Mandibular osteomyelitis
- Temporomandibular joint syndrome
- Migraine / cluster headache
- Temporal arteritis
- Post-herpetic neuralgia

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[12] American Academy of Neurology (2008 – reviewed 2021) *Diagnostic evaluation and treatment of trigeminal neuralgia*

# Motor neurone disease

Degenerative disease of UMNs & LMNs in the spinal cord & cortex

**Life expectancy significantly shortened**
(6 months to 3 years from onset of symptoms)

## General symptoms

- **Progressive weakness** but **no sensory deficit** (dysphagia, dysphonia, breathing difficulties)
- Mixed UMN &/or LMN signs (spasticity/fasciculations)

## Investigations

→ *clinical diagnosis*

→ **EMG:** active denervation, chronic neurogenic changes
→ **Bloods:** r/o other causes (CK, B12, folate, paraneoplastic Ab)
→ **Spinal MRI:** r/o myelopathy/radiculopathy

## Management[13]

**Supportive & palliative:** MDT approach

1. Respiratory support – consider NIV
2. Nutritional support (SALT) ± PEG tube
3. Muscle cramps – quinine, baclofen
4. Disease-modifying medication: riluzole – slows progression
5. Carer assessment & psychological support
6. End-of-life care plan

## Subtypes

| | **Amyotrophic lateral sclerosis (ALS)**<br>Most common | **Progressive muscular atrophy**<br>10% of cases | **Progressive bulbar palsy**<br>20% of cases | **Primary lateral sclerosis**<br>Rare |
|---|---|---|---|---|
| **Nerves affected** | **UMN & LMN** | **LMN** | **UMN & LMN** | **UMN** |
| **Pattern of weakness** | **Focal muscle weakness**<br>e.g. in one hand → gradual spreading to limbs/trunk | **Limb weakness**<br>Gradually spreads distal → proximal | **Dysarthria, dysphagia**<br>(choking/regurgitation) | Slowly progressing **tetraparesis** |
| **Associated signs/symptoms** | Fasciculations<br>+ spastic paresis<br>± fronto-temporal dementia | Fasciculations<br>+ painless wasting | Tongue fasciculations | + Pseudobulbar palsy |

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[13] NICE (2016, updated 2019) *Motor neurone disease* [NG42]

# Myasthenia gravis

An autoimmune disorder of the neuromuscular junctions. Pathogenesis: **autoantibodies** against the post-synaptic ACh receptor **block neuromuscular junction**

## Symptoms

**Weakness & fatiguability → worsens throughout day**

- Of proximal limbs – limits walking distance
- Of bulbar muscles – dysphagia/dysarthria
- Of ocular muscles – fluctuating diplopia & ptosis **(worsens with prolonged looking up)** → MG can be purely ocular
- Of respiratory muscles – breathing difficulties

**No UMN/LMN signs**

## Investigations

1. **Thorough history**
2. **Examination:** fatiguable tasks, full PNS & CNS exam
3. **Serum antibodies:** anti-ACh receptor, anti-Musk
4. **EMG: repeated nerve stimulation** → decreasing response
5. **Imaging:** CT for thymus masses

can be antibody negative

EMG can be DIAGNOSTIC

## Management[14]

1. **Oral anticholinesterases:** prolong ACh action (↓ breakdown) e.g. pyridostigmine
2. **Immunosuppression:** steroids ± disease-modifying drugs (azathioprine, methotrexate, mycophenolate)
3. **Thymectomy:** if hyperplasia/thymoma

**Acute crisis:** sudden worsening of weakness (± respiratory muscles)

→ **Rapid** plasmapheresis &/or IV immunoglobulins
→ ± Ventilation
→ ± NG feeding

**Associated with other autoimmune conditions**

- 25% also have thymoma
- 50–80% also have thymic hyperplasia

**Monitoring of MG:**

**Course** = lifelong & fluctuating

**Routine monitoring:** 4-monthly as an outpatient
**Monitoring acute flare:** regular forced vital capacity (FVC)

**Lambert–Eaton myasthenic syndrome**
Pre-synaptic NMJ disorder associated with small cell carcinoma of the lung

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[14] Sussman, *et al.* (2015) Myasthenia gravis: Association of British Neurologists' management guidelines. *Pract Neurol,* 15:199.

# Myopathies

= muscular disorder/weakness

↳
- **Myositis:** inflammatory disease
- **Muscular dystrophy:** inherited disorder of protein dysfunction
- **Channelopathies:** ion channel disorder of muscle

## General features

- **Symmetrical weakness**
- **Muscle wasting or pseudohypertrophy**
- Muscle pain/aches/cramps

± ptosis, ataxia, dysphagia, dysarthria, cardiac signs

**Categories of muscle disease:**

- Inflammatory
- Hereditary
- Metabolic
- Drug-induced

## General investigations

1. **Thorough Hx & clinical examination:** incl. FHx
2. **Serum CK:** raised
3. **EMG:** myopathic features
4. **Muscle MRI/biopsy:** shows inflammation / muscle changes
5. **Neurogenetic tests**

## Muscular dystrophies

### DUCHENNE & BECKER: X-linked recessive

| | Duchenne | Becker |
|---|---|---|
| **Onset** | From 5y | From 10y |
| **Symptoms** | • Progressive proximal weakness<br>• Calf pseudohypertrophy<br>• Waddling / slow running<br>• Gower's sign<br>• 30% = intellectual impairment | • Milder phenotype<br>• Not associated with intellectual impairment |
| **Prognosis** | Early 20s (respiratory failure / cardiomyopathy) | Late 40s → normal |

### MYOTONIC DYSTROPHY: most common dystrophy → autosomal dominant

**Symptoms:**

- **Myotonic grip:** slow to let go / open fist
- + distal muscle weakness
- + ptosis & facial muscle weakness
- ± respiratory & cardiac complications

**Differentiating myopathic & neurogenic cause of weakness**

| | Neurogenic | Myopathic |
|---|---|---|
| **Weakness** | Distal or focal | Proximal or bulbar |
| **Muscle pain / fatigue** | – | Pain in myositis<br>Fatigue in myasthenia |
| **Sensory loss / burning** | + | – |
| **Reflex loss** | + | – |
| **CK** | ↑ / normal | ↑↑↑ |
| **Fasciculation** | In MND<br>Not in peripheral neuropathy | – |
| **Clues in Hx** | Diabetes Sx<br>Viral Sx (GBS) | Often FHx |

# Essential tremor

Involuntary, rhythmical shaking movement most commonly affecting the upper limbs

## Clinical presentation

1. **Progressive tremor**
   - Bilateral & symmetrical (4–12Hz frequency)
   - Usually affects hands/arms, but can also affect voice & head
   - Present **during action & when holding a position** (postural)
   - Not usually present at rest (but can be when very severe)
   - Improves with alcohol
2. **No associated neurological symptoms** e.g. dystonia, ataxia, or parkinsonism

Tremor may impact fine motor control: writing, eating, drinking etc.

**Risk factors for essential tremor:**
- Increasing age
- FHx

## Investigations

essential tremor is a clinical diagnosis

- Examination – including Archimedes' spiral
- CT/MRI brain – if associated neurological symptoms
- TFTs – r/o hyperthyroidism
- DAT scan – if tremor is particularly asymmetrical &/or signs of Parkinson's disease

**Archimedes' spiral:** by asking a patient to draw this spiral, the frequency, amplitude & direction of tremor can be assessed

## Management[15]

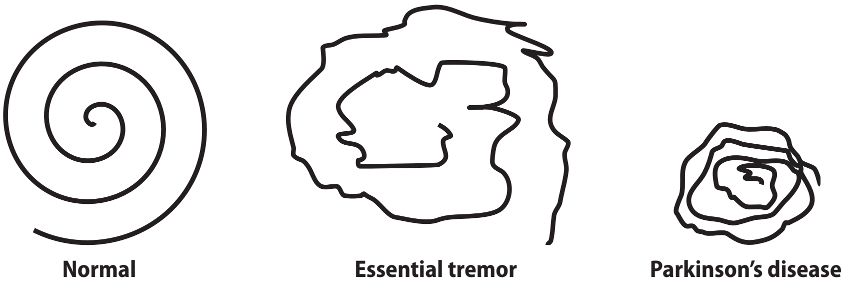
**No psychological or functional impact:** no intervention indicated

**Psychological or functional impact:**
- **1st line:** propranolol or primidone
- **2nd line:** gabapentin or topiramate

**Treatment-resistant tremor:** deep brain stimulation (DBS) → has associated complications/risks

**Differentials of essential tremor:**
- Parkinson's disease
- Dystonic tremor
- Psychogenic tremor
- Drug-induced tremor

(lithium, antipsychotics, caffeine)



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[15] BMJ Best Practice (2021) *Essential Tremor*

# Headaches

**Describing headaches:**

**S:** Site
**O:** Onset
**C:** Characteristic
**R:** Radiation
**A:** Associations
**T:** Timing
**E:** Exacerbating factors
**S:** Severity

## Classifying headaches

| Primary headaches | Secondary headaches |
|---|---|
| Migraine<br>Tension / medication overuse<br>Cluster | Subarachnoid haemorrhage<br>Raised ICP<br>Infection<br>GCA |

## Benign/primary headaches

| | Migraine | Cluster headache | Tension |
|---|---|---|---|
| **Symptoms** | **S: Unilateral**/bilateral<br>**O:** Hours (± worsening aura)<br>**C:** Throbbing<br>**A:** Photo-/phonophobia, N&V<br>**T:** 24–72h<br>**E:** Movement<br>Typically, aura develops from visual → sensory → motor | **S:** Above eye (unilateral)<br>**O:** Sudden<br>**C:** Severe<br>**A:** Tearing, nose running, sweating, conjunctival injection<br>**T:** 15–180min & recurs 1–8 × a day<br>**Relief:** Pacing (have restlessness) | **S: Frontal bilateral** 'band'<br>**O:** With stress/tiredness<br>**C:** Tightness |
| **Who?** | 10% of population<br>F:M = 3:1 | M:F = 3:1 | 30–70% of the population |
| **Risk factors / associations** | **Menstruation/OCP**<br>Physical exercise<br>Emotional stress<br>Foods (cheese, chocolate, red wine) | **Alcohol**<br>**Smoking** | **Medication overuse**<br>**Stress**<br>Depression |
| **Management**[14] | **r/o other differentials:**<br>e.g. raised ICP, SAH, meningism<br>**1st line:**<br>Lie in dark room **plus** oral NSAIDs/paracetamol<br>± anti-emetic<br>± 900mg soluble aspirin (avoid in asthmatics)<br>**2nd line:** triptans<br>**Prophylaxis:** if ≥4 a month or ≥1 a week, topiramate or propranolol | **r/o other causes of eye pain:**<br>e.g. acute glaucoma / GCA<br>**At start of attack:** SC triptan<br>**During attack:** home $O_2$ via non-rebreathe mask<br>**Prophylaxis:**<br>→ alcohol avoidance<br>→ verapamil/topiramate/prednisolone | **Episodic:** <15d/m<br>Paracetamol & NSAIDs<br>Advise on medication overuse<br>**If use medications >2d/w**<br>Low dose amitriptyline (start at 10mg and slowly ↑)<br>**Chronic:** >15d/m<br>Exclude medication overuse<br>Exclude depression<br>**Reassurance, relaxation** |

## Secondary headaches

### THUNDERCLAP HEADACHE

**Symptoms:**

**S:** Back of head / occipital
**O:** Sudden, peaks intensity in <1min
**C:** Like a blow to the head
**A:** Vomiting, neck stiffness
± subhyaloid (intraocular) haemorrhage

**Investigations: urgent CT**

**If CT is negative = lumbar puncture**
→ Look for xanthochromia in CSF
→ Must be ≥12h after headache onset but not useful after 2–3w

**Differentials of thunderclap headache:**

- Migraine
- Meningitis
- Coital headache – much briefer (30–60min) & recurrent
- Venous sinus thrombosis
- Reversible vasoconstriction syndrome
  → following steroids / nasal decongestants
  → Dx with CT angiogram

SAH until proven otherwise

### RAISED ICP HEADACHE

**Symptoms:**

**S:** Whole head / occipital area
**O:** Days to weeks
**A:** Papilloedema, focal Sx if mass lesion, transient visual obscuration
**T:** Progressively worsening
**E:** Bending, coughing, exertion, in mornings

**Investigations: urgent CT/MRI & CT/MRI venograms**
→ Check BP
→ If CT/MRI normal, LP to check opening pressure

**Management:** urgent neurosurgical referral

**Differentials/causes of raised ICP headache:**

- Mass lesion
- Hydrocephalus
- Venous sinus thrombosis
- Idiopathic intracranial hypertension (IIH)

Risk factors for IIH: female, ↑ BMI, tetracycline, OCP

### LOW PRESSURE HEADACHE

**Cause:** spinal CSF leak (can be due to epidural / spinal tap / penetrating wound, trauma, spontaneous)

**Symptoms:**

**S:** Whole head / occipital area
**O:** Days to weeks (can pinpoint starting day)
**A:** Neck pain, nausea, hearing change, imbalance
**T:** Progressively worsening
**E:** Being upright (absent when supine)

**Investigations: urgent CT/MRI head & spine**

**Management:**

**1st line:** bed rest + fluids + caffeine
**2nd line:** blood patch

### GIANT CELL ARTERITIS HEADACHE

**Symptoms:**

**S:** Temporal/occipital
**O:** Acute
**A:** Jaw claudication, scalp tenderness, polymyalgia rheumatica, weight loss, **vision changes**

tends to affect those >50y

**Investigations: urgent as risk of blindness**
→ CRP/ESR
→ Temporal artery biopsy

**Management:** high dose prednisolone + ophthalmology referral if eye symptoms

**Differentials of GCA:**

- Migraine
- Stroke

# 08

# RENAL

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## ABBREVIATIONS

**ACEi** – Angiotensin-converting enzyme inhibitor
**ACR** – Albumin–creatinine ratio
**AKI** – Acute kidney injury
**ANA** – Antinuclear antibody
**ANCA** – Antineutrophil cytoplasmic antibody
**ARB** – Angiotensin receptor blocker
**ATN** – Acute tubular necrosis
**BM** – Basement membrane
**BPH** – Benign prostatic hyperplasia
**Ca** – Calcium
**CK** – Creatine kinase
**CKD** – Chronic kidney disease
**Cr** – Creatinine
**CT KUB** – CT kidneys, ureters, bladder
**DM** – Diabetes mellitus
**DRE** – Digital rectal exam
**(e)GFR** – (estimated) Glomerular filtration rate
**HSP** – Henoch–Schönlein purpura
**HUS** – Haemolytic uraemic syndrome
**Ig** – Immunoglobulin
**MMF** – Mycophenolate mofetil
**Na** – Sodium
**Osm** – Osmolality
**PPI** – Proton pump inhibitor
**PSA** – Prostate-specific antigen
**PTH** – Parathyroid hormone
**RCC** – Renal cell carcinoma
**RRT** – Renal replacement therapy
**SLE** – Systemic lupus erythematosus
**TCC** – Transitional cell carcinoma
**UTI** – Urinary tract infection

# Acute kidney injury

= Rapid deterioration in kidney function over hours to days (= ↑ Cr)

**Incidence: common**
10–20% hospital admissions
20–30% cardiac surgery patients
50% ICU admissions

→ Address RFs in hospitalised patients to prevent AKI

→ **AKI = marker for mortality**

## KDIGO staging of AKI[1]

→ *based on **serum creatinine** (Cr) & **urine output***
→ *need baseline renal function*

| Stage | Serum creatinine | Urine output |
|---|---|---|
| 1 | 1.5–1.9× baseline* **OR**<br>>26.5µmol/L within 48h | <0.5ml/kg/h for 6–12h |
| 2 | 2–2.9× baseline | <0.5ml/kg/h for >12h |
| 3 | >3× baseline **OR**<br>>353µmol/L **OR**<br>requiring renal replacement therapy | <0.3ml/kg/h for >24h<br>**OR**<br>Anuric for >12h |

**Baseline Cr** = usual value over pre-morbid period

## Classification of AKI

| | **Pre-renal**<br>**Hospital = 70%**<br>**Community = 30%** | **Intra-renal**<br>**Hospital = 20%**<br>**Community = 25%** | **Post-renal**<br>**Hospital = 10%**<br>**Community = 40%** |
|---|---|---|---|
| **Causes** | **Volume depletion:**<br>• Blood loss / dehydration / burns<br>**Reduced BP:**<br>• Shock / cardiogenic shock<br>• Medications<br>**Reduced vascular perfusion:**<br>• Renal artery stenosis<br>**Renal vasoconstriction:**<br>• ACEi/NSAIDs | **Vascular:**<br>• Vasculitis<br>• HUS<br>**Glomerular:** glomerulonephritis (e.g. immune-mediated vasculitis)<br>**Tubular disease:**<br>• ATN (if persistent ↓ perfusion)<br>• Multiple myeloma<br>• Rhabdomyolysis<br>**Interstitial nephritis:**<br>• New drug (hypersensitivity) | **Intrinsic obstruction:**<br>• Malignancy<br>• Stones<br>• Stricture<br>**Extrinsic compression:**<br>• BPH<br>• Pelvic malignancy<br>• Retroperitoneal fibrosis<br>**Acquired:**<br>• Blocked catheter |
| **Urine dip** | No blood, no protein | Blood ± protein | Blood or leucocytes<br>No protein |
| **Urine osmolality (osm)** | **Can retain Na to concentrate urine**<br>= urine osm high, urine Na low | **Cannot retain Na / concentrate urine**<br>= urine osm low, urine Na high | **Cannot concentrate urine if kidney damage**<br>= urine osm low/normal |
| **Symptoms** | **Vague & non-specific → arise due to biochemical disturbances**<br>• Oliguria/anuria • Abdo pain • Dizziness, headache • Nausea, vomiting | | |
| | ± Signs of dehydration<br>± Signs of shock | ± Signs of vasculitis (rash etc.) | ± Abdo distension |
| **Management[2]** | 1. **ABCDE** – ABX if septic<br>2. **Stop nephrotoxic drugs**<br>3. **Assess & manage fluid status** – BP, pulse, HR, mucous membranes<br>4. **Assess & manage electrolyte imbalances** – daily U&Es, ECG | | **Regular monitoring:**<br>U&Es, BP, ABG |
| | Volume replacement (IV fluids) if volume depletion | Consider fluid restriction & loop diuretic if overloaded | 1. Consider urine drainage/catheter<br>2. **Refer to urology** |

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[1] KDIGO (2012) Clinical Practice Guideline for Acute Kidney Injury. *Kidney Intl*, 2(Suppl 1)
[2] NICE (2019) *Acute kidney injury* [NG148]

## Risk factors for AKI

- **Underlying conditions:** CKD, heart failure, liver disease, DM, cognitive impairment
- **Acute conditions:** sepsis, cardiac surgery
- **Medications:** **ACEis, NSAIDs,** PPIs, **gentamicin,** ciprofloxacin, allopurinol, penicillins, **lithium, contrast medium**
- **Dehydration:** elderly, kept NBM

## Complications of AKI

- Hyperkalaemia
- Fluid overload
- Pulmonary oedema
- Metabolic acidosis
- Uraemia

**Indications for dialysis:** Life-threatening, refractory AKI with complications
- Severe hyperkalaemia/acidaemia/uraemia
- Refractory pulmonary oedema
- Toxins/drugs

## Investigations[3]

1. **History & examination**
→ Risk factors for AKI → 'VENUS'
→ Symptoms suspicious of glomerulonephritis/vasculitis
→ DRE for enlarged prostate

2. **Urine dip** to r/o UTI & determine if underlying glomerular disease
→ if no blood or protein – **pre-renal** or **tubulointerstitial cause**
→ if blood & protein – **renal cause**
→ if blood only – **post-renal** or **renal cause**
→ if nitrites ± leukocytes – need to r/o infection → **MCS**

3. **Further urinalysis:** MCS, casts, Bence Jones protein (if suspect myeloma e.g. high calcium)

4. **Bloods: need to know baseline** (review previous creatinine)
→ FBC – raised CRP/ESR if infection/inflammation
→ U&Es – raised Cr, urea, $K^+$, $Na^+$
→ **nephritic screen** – if unexplained AKI/CKD **or** features suggesting vasculitis

5. **Arterial blood gas** – if severe **acidosis**, consider **overdose**

6. **ECG** – signs of hyperkalaemia (flat P, broad QRS, tented T)

7. **Renal USS or CT KUB** – may show signs of obstruction (hydronephrosis, distended bladder, stones)

**Severe hyperkalaemia** (>6mmol/L)

**Symptoms:**
Chest pain, palpitations, dizziness

**Investigations:**
U&Es, fluid balance, ECG

**Management:**
1. 10ml 10% calcium gluconate = stabilise myocardium
2. IV insulin + 25g glucose = drive potassium into cells
3. Salbutamol nebs = drive potassium into cells
4. 1.4% sodium bicarbonate = correct acidosis
5. Treat cause

**V**olume depletion

**E**xtra history
- **H**aemolysis, **h**aemoptysis, **h**ypercalcaemia
- **R**ash, **r**aised CK, **r**ecent vascular intervention

**N**ephrotoxics (medication review)

**U**rinary symptoms (voiding, haematuria, oliguria)

**S**epsis

**Nephritic screen** – tests for intrarenal causes
- ANCA (vasculitis)
- ANA/ds DNA (SLE)
- Complement (low in infection/SLE)
- Hepatitis B and C serology

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[3] Nitin, *et al*. (2016) A simple care bundle for use in acute kidney injury: a propensity score-matched cohort study, *Nephrol Dial Transplant*, 31:1846

# Chronic kidney disease

= progressive & irreversible deterioration of renal function (>3m)

### Factors increasing risk of needing RRT

- Younger age
- Lower eGFR
- Male gender
- Higher ACR

### Signs/symptoms:

Early stages = **asymptomatic**

- **Anaemia:** pallor, fatigue
- **Bone disease:** osteomalacia/fractures
- **Hyperkalaemia:** palpitations, dizziness, chest pain
- **Fluid overload:** peripheral oedema, SOB
- **Early uraemia:** fatigue, anorexia, N&V, pruritus, hiccups
- **Late uraemia:** confusion
- **Metabolic acidosis:** ↑ HR & RR, vomiting, fatigue, headache
- **Other:** sexual dysfunction = common

** CKD associates with cardiovascular events & AKI **

### Investigations:

1. **Urine dip & BP**
   → Urine protein–creatinine ratio for nephrotic syndromes
   → Urine albumin–creatinine ratio (ACR) for CKD
2. **Bloods:**
   → U&Es – **compare to previous eGFR**
   → Bicarbonate – acid–base balance lost
   → Hb – normocytic anaemia
   → PTH (↑ if bone disease & also ↑ ALP)
   → Glucose (for DM)
   → ANA, ANCA, complement
3. **USS kidney** (kidneys may be small)
4. **Renal biopsy** – if still unsure of cause
5. **CXR** – for pulmonary oedema

### Pathogenesis of renal bone disease:

↓ **Vit D activation** in kidneys = ↓ Ca absorption = **2° hyperparathyroidism** (↑ PTH)

↑ PTH causes ↑ **osteoclastic activity** = ↑ Ca resorption from bone to restore serum Ca = **leaves bone weak**

In kidney transplant patients, parathyroid gland may become autonomous in spite of normal renal function = **tertiary hyperparathyroidism**

## Classification

→ *use to identify those most at risk of needing RRT*

**Base on 2 factors:**

1. GFR
2. Albuminuria

| | | | Persistent albuminuria | | |
|---|---|---|---|---|---|
| | | | **A1** | **A2** | **A3** |
| | | | <3mg/mmol | 3–30mg/mmol | >30mg/mmol |
| **GFR** ($ml/min/1.73m^2$) | **G1** | ≥90 | 🟩 | 🟨 | 🟧 |
| | **G2** | 60–89 | 🟩 | 🟨 | 🟧 |
| | **G3a** | 45–59 | 🟨 | 🟧 | 🟥 |
| | **G3b** | 30–44 | 🟧 | 🟥 | 🟥 |
| | **G4** | 15–29 | 🟥 | 🟥 | 🟥 |
| | **G5** | <15 | 🟥 | 🟥 | 🟥 |

Table based on KDIGO AKI staging system.

## Causes of deteriorating GFR

| | |
|---|---|
| **Diabetes mellitus** (38%) | Glycation of efferent arteriole = ↑ pressure & sclerosis |
| **Hypertension** (25%) | Thickened walls of afferent arteriole = hypoperfusion |
| **Chronic glomerulonephritis** (16%) | Inflammation & damage to vessels |
| **Chronic pyelonephritis** | Urinary reflux or recurrent infection |
| **Obstructive uropathy** | Back-up of pressure = hydronephrosis & damage<br>→ neurogenic bladder, BPH, malignancy, stones |
| **Polycystic kidney disease (PKD)** | → **Auto dominant** form presents in adults<br>→ Fluid-filled cysts press on nephrons = atrophy<br>→ Sx: back pain, headaches, haematuria, HTN |

→ **Risk factors for CKD decline:** HTN, DM, smoking, infection, NSAIDs/ACEis

## Management of CKD[4]

**RISK REDUCTION of CVD:** lifestyle factors are important

1. **BP control:** ACEi/ARB if proteinuria (BP<140/90 **or** <130/80 if diabetic)
2. **Cholesterol control:** statin
3. **Comorbidity control:** diabetes control
4. **Stop smoking**
5. **Weight management**

Advise low salt & phosphate diet

**TREAT/MANAGE COMPLICATIONS**

1. **Anaemia** – IV iron + EPO stimulating agents (r/o B12/folate deficiency first)
2. **Bone disease** – vit D & calcium supplements if deficient, phosphate binders
3. **Oedema** – careful fluid monitoring ± diuretics

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[4] NICE (2021) *Chronic kidney disease* [NG203]

### MANAGE MEDICATIONS

1. **Stop drugs** that worsen glomerular function / acute nephrotoxics
2. **Alter dose** of medications if GFR is low e.g. stop metformin if GFR <30

**REFER TO NEPHROLOGY:** if GFR <30 or ↓ >15 in 1y **or** 5y risk of needing RRT >5% (using kidney failure risk equation[5])

→ assess & manage symptoms/complications & prep for RRT

## Renal replacement therapy options

ensure patients are fully informed of all options & supported in decision-making

**DIALYSIS:** usually started around eGFR 10, unless there are complications

| | Haemodialysis | Peritoneal dialysis |
|---|---|---|
| **Method** | Blood pumped out of body through 'artificial kidney' | Dialysate solution infused into peritoneal cavity (peritoneum acts as filtering membrane) |
| **Access** | AV fistula / semi-permanent jugular or subclavian catheter | Catheter into peritoneum |
| **Complications** | Site infection, **hypotension**, air embolus, N&V, endocarditis | Peritonitis, catheter problems, hernia, fluid retention, weight gain |
| **Frequency** | 3 × 4h sessions each week | **Continuous ambulatory** = 4 × 20min each day while active<br>**Automated** = overnight (3–5 exchanges over 8–10h) |

**KIDNEY TRANSPLANT:** **gold standard** for those with **end-stage CKD**

→ **but** only 40% of patients with CKD 5 are suitable

**Process:**

→ Transplant placed in **iliac fossa** & anastomoses of vessels made

→ Usually leave native kidney in place

**Post-transplant treatment:**

→ Lifelong immunosuppression = tacrolimus or ciclosporin **plus** azathioprine/MMF

→ 6m of steroids to prevent acute rejection e.g. prednisone

→ Aspirin, antihypertensives, PPI, bone protection

**Complications:**

- **Immediate:** graft thrombosis, ureteric leak/obstruction, bleeding, rejection
- **3–6m:** rejection, HTN, ileus, infections (urinary or respiratory)
- **Long-term:** cancer, interstitial fibrosis (2° to ciclosporin/tacrolimus), cardiac disease, infections

**Ethical issue:** waiting list of >4500 (avg. wait = 3y)

**Points to consider for transplant:**

| Pros | Cons |
|---|---|
| ↑ Longevity (80% 10y survival)<br>↑ Quality of life (free from dialysis) | Medication burden<br>Immunosuppressive SEs<br>Frequent hospital visits |

**Who is suitable?**

- Generally fit for general anaesthetic
- At least 5y left to live
- No underlying malignancy
- No other significant comorbidities
- Good vascular supply to legs

**+ a suitable match is found:**

→ ABO, tissue type, age & gender

**+ Need to assess suitability of donor:** urine dip, U&Es, USS & general fitness

**Transplant rejection:** 10–20%

→ **Often asymptomatic** (concern if rise in Cr)

→ **RFs:** non-concordance, drug interactions, poor match

→ **Tx:** IV methylprednisolone & ↑ immunosuppressants

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[5] Major, *et al.* (2019) The Kidney Failure Risk Equation for prediction of end stage renal disease in UK primary care. *PLOS Medicine*, 16:e1002955

# Glomerular disease

## Nephrotic syndrome

→ *inflamed basement membrane (BM) (damaged podocytes) allows passage of proteins into nephron*

↳
1. **Heavy proteinuria** (>3.5g/d)
2. **Hypoalbuminaemia** (<25g/L)
3. **Oedema** – periorbital, legs, scrotal/vaginal

can also get haematuria if lots of BM damage

patients often report FROTHY URINE

### CLINICAL MANIFESTATIONS:

- Peripheral & periorbital oedema (loss of albumin)
  - → ± ascites, abdo pain & leukonychia
- Pleural effusion (loss of albumin)
  - → SOB/orthopnoea
- Hyperlipidaemia (loss of liporegulatory proteins)
  - → ± xanthelasma & xanthoma
- Hypercoagulability (loss of thromboregulatory proteins)
- Infections (loss of immunoglobulins)

### CAUSES:

| **Primary**<br>→ immune complex deposition &/or complement activation | **Secondary**<br>→ damage due to underlying disease |
|---|---|
| 1. **Minimal change disease** = **most common in children** | 1. **Diabetic nephropathy** = **most common cause** |
| • microscopically normal renal tissue | 2. **SLE** |
| 2. **Focal segmental glomerulonephritis** | 3. **Infection:** hepatitis B/C, HIV, malaria, syphilis |
| • thickened BM & focal areas of sclerosis | 4. **Drugs:** penicillamine, gold, NSAIDs, iron |
| • results in damage to podocytes | 5. **Amyloidosis** |
| 3. **Membranous glomerulonephritis** = **30% adult cases** | 6. **Myeloma** |
| • complement activation & IC deposition causes BM thickening & podocyte damage | 7. **Pre-eclampsia** |
| • (anti-phospholipase a2 antibodies present) | |

### INVESTIGATIONS:

**Confirming diagnosis**

- Urine dip – protein
- Urine MCS – r/o UTI
- U&Es, Cr, albumin
- FBC, ESR/CRP, LFTs
- **Renal biopsy** – shows microscopic changes to glomeruli (confirms subtype)

**Determine cause:**

- Autoimmune screen (ANCA, anti-phospholipase a2, complement levels)
- Serum & urine immunoglobulins / electrophoresis
- Infection screen (throat swab / anti-strep, hep B & C, HIV, syphilis, malaria)

**Assess complications:**

- CXR – pleural effusion
- Lipid profile – loss of liporegulators in urine
- TFTs – loss of thyroid hormones in urine
- Coag screen – loss of thromboregulators in urine

### MANAGEMENT[6]: nephrologist input

1. **Reduce oedema:** loop diuretics + fluid & salt restriction (max 1L fluid/d)
2. **Reduce proteinuria:** ACEis/ARBs (cause efferent vasodilation)
3. **Treat underlying cause:** corticosteroids indicated in minimal change disease

aim for 0.5–1kg weight loss per day

---

[6] KDIGO (2021) *Clinical Practice Guideline for the Management of Glomerular Diseases*

## Nephritic syndrome

→ *specific form of glomerulonephritis*

→ Inflammation **damages glomerular capillary** so **RBCs leak** into nephron → RBCs clump together = casts

→ Inflammation **damages podocytes** allowing **protein to leak** into nephron

→ Inflammation causes **reduced GFR**, leading to **oliguria & hypertension**

↳
1. **Haematuria** (>10 red cell casts)
2. **Oliguria** (<0.5–1ml/kg/h)
3. **Proteinuria**

### CLINICAL MANIFESTATIONS:

- **Volume overload / reduced GFR**
  - Oedema (esp. periorbital)
  - Hypertension → seizures
- **If severe presents as AKI:** oliguria

### CAUSES:

1. **Autoantigens (Goodpasture's disease):** anti-BM antibodies (often also get alveolar haemorrhage & haemoptysis)
2. **ANCA-associated (small vessel vasculitis):** *p*-ANCA & *c*-ANCA activate macrophages & neutrophils = immune damage
   - Prodrome of malaise, arthralgia/myalgia, skin rash & AKI
   - **No immune complex deposition seen**
3. **Immune complexes:** immune complex deposits activate complement & inflammation
   - Results in low complement
   - **Causes*:** IgA nephropathy, post-strep infection (HSP) & SLE

*Any of these can lead to **rapidly progressing glomerulonephritis & acute renal failure**

### INVESTIGATIONS: same as for nephrotic syndrome

- Often low complement
- Check for ANCA, ANA & anti-strep

### MANAGEMENT:

1. **Goodpasture's disease:** plasmapheresis (removes Abs), corticosteroids, cyclophosphamide (immunosuppressant)
2. **ANCA-associated vasculitis:** induce remission with corticosteroids & cyclophosphamide → maintain with azathioprine
3. **IgA nephropathy / post-strep infection:** ACEi/ARB (protect renal function), ± corticosteroids

**Other Mx:**

- **Diuretics if nephrotic component** – maintain fluid & electrolyte balance
- **Treat infection if present** – ABX (penicillin)
- **If rapid ↓ renal function:** renal biopsy, immunosuppression, plasma exchange

### GLOMERULONEPHRITIS:

**Definition:** name for the group of diseases causing **immune-mediated inflammation** of glomerulus (= ↑ permeability)
= **Spectrum of nephrotic & nephritic syndromes**

**Causes:**

- Post-infections (malaria/staph/salmonella/strep)
- Systemic autoimmune diseases / vasculitis e.g. SLE

**Symptoms:**

- Proteinuria & haematuria
- Features of systemic autoimmune vasculitis

(sweats, weight loss, arthralgia, eye signs, rash, mouth ulcers)

**Treatment:**

- Diuretics ± steroids ± ABX

# Urinary tract infection

→ very common (F>M)

**Risk factors for UTI:**
- Female
- Sexual intercourse
- Pregnancy
- Diabetes
- Dehydration
- Immunosuppression (e.g. ABX)
- Incomplete emptying
- Structural abnormality / reflux
- Instrumentation (e.g. catheter)

**Common organisms:**
1. *E. coli* = 85%
2. *Staph. saprophyticus* = 5–10% ↳ in young / sexually active
3. Enterococci
4. Proteus
5. Klebsiella
6. Pseudomonas if catheter

**DO NOT** urine dip if >65y, send straight for MCS

## Clinical presentation

| | Upper (pyelonephritis)[7] | Lower (cystitis)[8] |
|---|---|---|
| **Symptoms** | • Dysuria (burning)<br>• Frequency/urgency<br>• **Flank/loin** pain<br>• **High grade fever, N&V**<br>• ± Haematuria<br>**Complications:** sepsis, AKI, perinephric abscess, renal papillary necrosis (CKD if chronic) | • Dysuria (burning)<br>• Frequency/urgency<br>• **Suprapubic** pain<br>• Foul-smelling, cloudy urine<br>→ **Elderly:** confusion ± incontinence<br>→ **Young:** vague, irritability, poor feeding |
| **Investigations** | 1. **Urine dip:** leukocytes (sensitive), nitrites (specific)<br>2. **MSU for MCS:** confirm Dx ($>10^5$ WCC/ml)<br>3. **Bloods:** FBC, CRP/ESR, U&Es, culture (if systemically unwell / pyelonephritis)<br>4. **USS KUB:** if recurrent, Tx-resistant or male | |
| **Management** | 1. **Admit**<br>2. **Analgesia**<br>3. **IV ABX:** ciprofloxacin or cefuroxime (7d) | 1. **Lifestyle:** hydration, personal hygiene<br>2. **Analgesia:** NSAIDs, paracetamol<br>3. **ABX:** trimethoprim or nitrofurantoin<br>→ **Women:** 3d course → **Men:** 7d course |
| | **Do not treat asymptomatic bacteriuria** unless pregnant | |

Urinary tract TB: sterile pyuria with urinary Sx + fever, sweats, weight loss

# Haematuria

**Pseudo-haematuria:**
- Menstruation
- Dyes/food
- Drugs (rifampicin)

**Summary of causes of haematuria:**
- UTI
- Stones
- Trauma
- Malignancy
- BPH
- Iatrogenic

## Classification

- Microscopic or macroscopic
- Asymptomatic or symptomatic (associated with other urinary symptoms/pain)

## Causes

| Upper urinary tract | | Lower urinary tract | | |
|---|---|---|---|---|
| **Kidney** | **Ureter** | **Bladder** | **Prostate** | **Urethra** |
| • UTI<br>• Stones<br>• Trauma<br>• Cancer (RCC or TCC) | • Cancer (TCC)<br>• Stones | • UTI<br>• Stones<br>• Trauma<br>• Cancer (TCC)<br>• Iatrogenic (catheter) | • BPH<br>• Adenocarcinoma | • Cancer<br>• Trauma |

## Investigations

1. **Hx & exam:** remember DRE
2. **Urinalysis:** dip & MSU
3. **Bloods:** FBC, clotting, U&Es, PSA
4. **Renal USS:** show calculi
5. **Flexible cystoscopy:** show bladder/urethral abnormalities
6. **CT KUB:** gold standard → if suspicious of cancer / recurrent haematuria

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[7] NICE (2018) *Pyelonephritis (acute)* [NG111]

[8] NICE (2018) *Urinary tract infection (lower)* [NG109]

# 09

# RESPIRATORY

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## ABBREVIATIONS

**ABPA** – Allergic bronchopulmonary aspergillosis
**Ach** – Acetylcholine
**ACTH** – Adrenocorticotrophic hormone
**ADH** – Antidiuretic hormone
**BBs** – Beta-blockers
**BDR** – Bronchodilator reversibility
**BiPAP** – Bilevel positive airway pressure
**BNP** – B-type natriuretic peptide
**CAP** – Community-acquired pneumonia
**CF** – Cystic fibrosis
**CFTR** – CF transmembrane conductance regulator
**COPD** – Chronic obstructive pulmonary disease
**CPA** – Costophrenic angle
**CPAP** – Continuous positive airway pressure
**CTD** – Connective tissue disorder
**CWP** – Coal workers' pneumoconiosis
**FeNO** – Fraction of expired nitrous oxide
**FEV$_1$** – Forced expiratory volume in 1 second
**FVC** – Forced vital capacity
**GORD** – Gastro-oesophageal reflux disease
**HAP** – Hospital-acquired pneumonia
**ICS** – Inhaled corticosteroids
**ILD** – Interstitial lung disease
**JVP** – Jugular venous pressure
**LABA** – Long-acting beta agonist
**LAMA** – Long-acting muscarinic antagonist
**LDH** – Lactate dehydrogenase
**LN** – Lymph node
**LRTI** – Lower respiratory tract infection
**LTRA** – Leukotriene receptor antagonist
**MAB** – Monoclonal antibody
**MRC** – Medical Research Council
**MTX** – Methotrexate
**NIV** – Non-invasive ventilation
**NMD** – Neuromuscular disorder
**NO** – Nitrous oxide
**PCR** – Polymerase chain reaction
**PE** – Pulmonary embolism
**PEF** – Peak expiratory flow
**PEG** – Percutaneous endoscopic gastrostomy
**PGs** – Prostaglandins
**PLA$_2$** – Phospholipase A$_2$ receptor
**QoL** – Quality of life
**RA** – Rheumatoid arthritis
**RSV** – Respiratory syncytial virus
**RVF** – Right ventricular failure
**SABA** – Short-acting beta agonist
**SAMA** – Short-acting muscarinic antagonist
**SIADH** – Syndrome of inappropriate ADH secretion
**SLE** – Systemic lupus erythematosus
**SOB** – Shortness of breath
**SVC** – Superior vena cava
**T1RF** – Type 1 respiratory failure
**T2RF** – Type 2 respiratory failure
**TB** – Tuberculosis

# Respiratory failure

| | **Type 1 respiratory failure (hypoxic)** | **Type 2 respiratory failure (hypercapnic)** |
|---|---|---|
| **$PaO_2$** | <8kPa | <8kPa |
| **$PaCO_2$** | **Normal (or ↓)** | **High (>6kPa)** |
| **Symptoms** | **Hypoxia**<br>Tachypnoea, dyspnoea, tachycardia confusion, cyanosis | **Hypercapnia**<br>Headache, altered behaviour, coma papilloedema, warm extremities |
| **Causes** | **V/Q mismatch (failed oxygenation of blood)**<br>**1.** Poorly ventilated alveoli<br>**2.** Poor perfusion of alveoli | **Insufficient alveolar ventilation**<br>**1.** Increased airway resistance<br>**2.** Reduced respiratory drive |
| **Examples** | **1.** Pulmonary oedema, pneumonia, asthma<br>**2.** Pulmonary embolism | **1.** **↓TV:** COPD, chest deformities, obesity<br>**2.** **↓RR:** neuromuscular, sedatives, exhaustion |
| **Mx** | **1.** **For ↓ventilation:** high flow $O_2$ effective (94–98%)<br>**2.** **For ↓perfusion:** high flow $O_2$ not effective<br>**If $O_2$ not effective, consider ventilation:**<br>CPAP(NIV) or sedation + invasive ventilation (on ICU) | **1.** **Controlled flow $O_2$ & treat underlying cause** (ideally use device allowing fixed rate delivery of 88–92% e.g. Venturi mask)<br>**If $O_2$ not effective, consider ventilation:**<br>BiPAP (NIV) or sedation + invasive ventilation |

| **Restrictive lung disease** | **Obstructive lung disease** |
|---|---|
| **↓Lung capacity** but no ↓in ability to inspire/expire | Normal lung capacity but **↓in ability to inspire/expire** |
| **$FEV_1$** = decreased <80%<br>**FVC** = decreased <80%<br>**$FEV_1$:FVC** = normal >0.7 | **$FEV_1$** = decreased <80%<br>**FVC** = normal / small decrease >80%<br>**$FEV_1$:FVC** = decreased <0.7 |
| Obesity / pregnancy<br>Pulmonary fibrosis / pneumoconiosis<br>Pulmonary oedema<br>Lobectomy<br>Skeletal abnormalities<br>Neuromuscular disorders | Asthma<br>COPD/emphysema<br>Bronchiectasis |

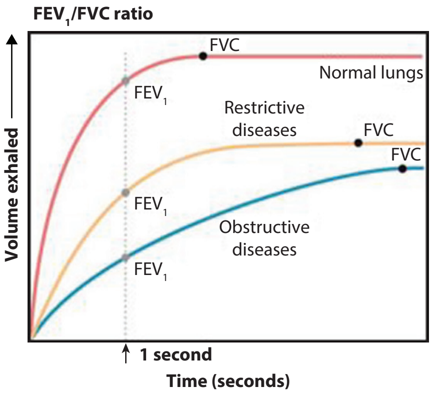

**Fig. 9.1** Spirometry interpretation.

# Pneumothorax

= air in the pleural space causing **lung collapse**

From rupture of blebs/bullae

| | **Spontaneous** | | **Iatrogenic** | **Traumatic** |
|---|---|---|---|---|
| **Cause** | **Primary (no lung disease)**<br>Male, tall & slim (Marfan)<br>Smoker | **Secondary to lung disease**<br>Pulmonary TB, pneumonia<br>COPD, emphysema, CF | Mechanical ventilation<br>Bronchoscopy<br>Central venous catheter | Blunt trauma<br>Penetrating wound |
| **Age** | 20–30y | 60–65y | Any | Any |
| **Sx** | May be asymptomatic<br>**Sudden onset breathlessness**<br>Unilateral pleuritic pain<br>Tension pneumothorax = clinical Dx<br>↓Breath sounds & expansion, hyper-resonance<br>+ tracheal deviation, severe resp. distress & shock **in tension pneumothorax** | | | |
| **Mx**[1] | **<2cm:** $O_2$ / discharge & f/u<br>**>2cm or SOB:** aspirate | **<1cm:** $O_2$ & monitor 24h<br>**1–2cm:** aspirate<br>**>2cm or SOB:** chest drain | **May not need Tx if no Sx** | **Urgent decompression**<br>Large-bore cannula<br>or chest drain |

**Investigations:**
1. CXR
2. ABG
3. CT

**ASPIRATION:**

→ Wide-bore cannula, 3-way tap & 50ml syringe

→ **2nd intercostal space, mid-clavicular line**

**CHEST DRAIN:**

→ Various types with seal

→ + High vol, low pressure suction if not resolving (drain still bubbling)

→ **5th intercostal space, mid-axillary line**

**SURGICAL OPTIONS:**

- **Pleurectomy/blebectomy**
- **Pleurodesis:** adhere lung to chest wall

**Indications for surgical Mx:**
- Not resolving despite chest drain & suction
- Recurrent pneumothorax

## Practicalities of chest drains

**TYPES:**

1. **Blunt dissection:** incision in skin + blunt dissection with fingers to insert plastic tube between 2 ribs → suture *in situ*
2. **Seldinger drain (3-way-tap):** needle & guidewire used to place tube → suture *in situ*
3. **Pigtail drain:** US/CT-guided placement → curled end prevents it dislodging
4. **Indwelling pleural catheter:** tunnelled under skin before entering pleural cavity → longer-term drainage

**Long term Mx of pneumothorax:**
- no flying 1w
- no deep-sea diving for life
- elective surgery if recurrent

**COMMON COMPLICATIONS:**

- Discomfort – prescribe codeine/paracetamol/NSAID
- Infection – insert with aseptic technique & check regularly
- Bleeding – insert in 'triangle of safety' above (not below) rib
- Incorrect placement – insert with US guidance if possible
- Blockage – inspect fluid in tube → should **'swing'** with inspiration/expiration if not blocked
- **Premature removal in pneumothorax** – inspect for **bubbling** at rest / with cough → indicates drain still needed

**Common indications for chest drains:**
- Tension / Tx-resistant pneumo/haemothorax
- Symptomatic pleural effusion

**WHAT TO REVIEW ON THE WARD:**

- **Insertion site:** drain twisted? infection?
- **Swinging:** if not suggests blockage / tube kinked
- **Bubbling:** in pneumothorax (if present = too early to remove)
- **Drainage chart:** record of swinging/bubbling/volume

→ If blocked **flush with 20ml saline** (via 3-way-tap)

→ In pleural effusion **shut tap after 1.5–2L for 4h** to avoid draining too quickly

→ never shut off in pneumothorax

---

[1] BMJ Best Practice (2021) *Pneumothorax*

# Pleural effusion

**Types of fluid**

**Empyema:** pus (infection)
**Chylothorax:** lymphatic fluid
**Haemothorax:** blood (trauma)
**Fluid:** transudate or exudate

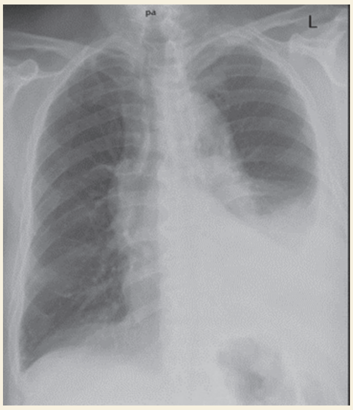

**Fig. 9.2** Left-sided pleural effusion.

Accumulation of fluid in pleural cavity

## Symptoms

**May be asymptomatic**

- SOB ± pleurisy
- Sx of underlying cause

## Signs

- ↓ Expansion
- Stony dull percussion
- Absent breath sounds
- ↓ Vocal resonance / tactile fremitus
- Tracheal deviation away if massive

## Investigations

1. **CXR** – blunted CPAs, homogenous consolidation (only detect if >300ml)
2. **USS** – identify location & volume
3. **Pleural tap (US guidance)***
   - Microbiology: MCS
   - Clinical chemistry: protein, LDH, glucose, pH
   - Cytology: 80% sensitive for malignancy
4. **Further Ix if no obvious cause:**
   - Pleural biopsy – r/o malignancy/TB
   - CT with contrast – shows pleural thickening

*1–2 intercostal spaces below upper border of effusion

## Types of pleural effusion

| | Transudate | Exudate |
|---|---|---|
| **Pathophysiology** | **Movement of fluid from circulation → pleural space**<br>↑ Capillary hydrostatic pressure **or**<br>↓ Capillary oncotic pressure | **Production & secretion of fluid into pleural space**<br>↑ Capillary permeability |
| **Causes** | • Congestive heart failure (↑ hydrostatic pressure)<br>• Renal failure (↓ oncotic pressure ↓ **albumin**)<br>• Liver failure (↓ oncotic pressure ↓ **albumin**)<br>• Hypothyroidism | **Inflammation:** trauma, RA, sarcoid, SLE<br>**Infection:** pneumonia, TB<br>**Infarction:** PE, post-MI (Dressler's)<br>**Malignancy:** 25% of effusions<br>**Medication:** MTX, amiodarone, phenytoin |
| **Presentation** | **Bilateral** | **Unilateral** |
| **Fluid** | **Lower protein, lower LDH**<br><25g/L protein + ↑ LDH | **Higher protein, higher LDH**<br>>30g/L protein + ↑↑ LDH |
| **Management** | **Usually improve with Tx of underlying cause**<br>→ Usually do not need tapping/drainage | **Pleural tap all with pneumonic illness or suspected malignancy + effusion**<br>Treat cause & drain if moderate/large |

### LIGHT'S CRITERIA

used to distinguish transudate & exudate if protein 25–35g/L

| | Transudate | Exudate |
|---|---|---|
| **Pleural:serum protein** | <0.5 | ≥0.5 |
| **Pleural:serum LDH** | <0.6 | ≥0.6 |
| **Pleural fluid LDH** | <2/3 upper limit of normal | >2/3 upper limit of normal |

## Specific management of pleural infection (empyema)[2]

1. **Pleural tap:** **pH <7.2, low glucose, high LDH**
   - Simple parapneumonic infection = straw colour with no orgs
   - Loculated empyema = pockets of semi-solid pus
2. **IV ABX** for minimum 2w
3. **Chest drainage:** if frank pus / organisms cultured / pH <7.2 / no improvement
4. **Decortication (VATS: video-assisted thoracoscopy):** remove restrictive layer of fibrous tissue → if long-standing pus / thickened pleura

Sx of empyema = effusion + FEVER

**Organisms causing empyema:**
*Strep. milleri, H. influenzae, E. coli, Staph. aureus, Pseudomonas*

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[2] BMJ Best Practice (2021) *Pleural effusion*

# Pneumonia

= signs of respiratory tract infection + new shadowing on CXR

| Community-acquired pneumonia | Hospital-acquired pneumonia |
|---|---|
| Primary or secondary to lung disease | **>48h** after admission or **within 10d** of discharge |
| • ***Strep. pneumoniae*** (80%)<br>• ***H. influenzae*** (more common in COPD)<br>• *Mycoplasma pneumoniae** (younger patients)<br>• *Chlamydia pneumoniae** (elderly patients)<br>• *Legionella pneumoniae** *Atypicals<br>• **Viral:** RSV, influenza, Covid-19 (15%) | • **Gram-negatives:**<br>► Klebsiella<br>► *E. coli*<br>► *Pseudomonas*<br>• MRSA / *Staph. aureus* |

**Aspiration pneumonia:** in stroke, neuromuscular disease, ↓ GCS

## Clinical presentation

- **Fever,** rigors, malaise, anorexia
- **Productive cough** – rusty sputum / haemoptysis
- **Dyspnoea**
- Pleuritic chest pain

## On examination

- **↑ RR, ↑ HR, ↑ temp**
- ↓ $O_2$ sats / cyanosis
- ↓ GCS / delirium – if elderly
- **Signs of consolidation**
  - **↓ expansion**
  - **coarse crackles**
  - pleural rub, bronchial breathing
  - **dull percussion**
  - ↑ vocal resonance

## Investigations[3]

- **Bedside:** basic obs, urine dip
- **Bloods:**
  - FBC, U&Es, CRP, LFT, glucose
  - ABG
  - Atypical serology
- **Microbiology:**
  - Sputum & blood cultures (guide ABX choice)
  - Urine antigens (atypical orgs)
  - Throat swab (if suspect viral)
- **Chest X-ray:** consolidation = **DIAGNOSTIC**

## Management[3]

1. **Conservative:** analgesia, $O_2$, antipyretics, IV fluids, chest physio
2. **Antibiotics:** follow local antimicrobial guideline (see table below for common examples)

| | | 1st line | If penicillin allergy | Duration & route |
|---|---|---|---|---|
| **CAP** | **Mild** | Amoxicillin 500mg TDS | Doxycycline | 5d PO |
| | **Mod** | Amoxicillin 500mg TDS + clarithromycin 500mg TDS | Doxycycline + clarithromycin | 7d PO |
| | **Severe** | Co-amoxiclav + clarithromycin | Levofloxacin + vancomycin | 10d IV |
| **HAP** | **Mild** | Doxycycline PO | | 5d PO |
| | **Severe** | Co-trimoxazole PO | | 5–7d IV |
| **Aspiration** | | Amoxicillin + **metronidazole** | | 5–7d IV |

3. **Long-term:** smoking cessation advice, influenza vaccine if high risk

**Risk factors for pneumonia:**
- Immunocompromised
- Hospitalised
- Chronic lung disease
- Elderly/young/male
- Alcoholic/smoker/IVDU

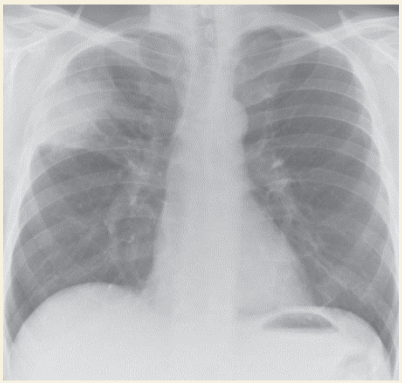

**Fig. 9.3** Right upper zone consolidation.

**Risk stratification:** CURB65 score

| | | |
|---|---|---|
| **C** | Confusion | MMSE ≤8 **or** disorientated |
| **U** | Urea | >7mmol/L |
| **R** | Resp rate | ≥30/min |
| **B** | BP | SBP <90 or DBP <60 |
| **65** | Age | >65y |

**Mild:** 0–1 = home Tx
**Mod:** 2 = hospital Tx
**Severe:** ≥3 = consider ICU

**Complications of pneumonia:**
- Pleural effusion
- Resp. failure
- **Sepsis**
- Empyema
- Lung abscess
- Bronchiectasis
- Pneumothorax
- Lobe collapse

**Repeat CXR after 6w** to ensure resolution & no underlying malignancy / lung abnormalities

---

[3] BMJ Best Practice (2021) *Community acquired pneumonia*

# Tuberculosis

Infection caused by **mycobacterium** (*M. tuberculosis, M. bovis, M. africanum*)

Estimated 1/4 of world infected → many of those have HIV (most new cases are in developing countries)

**Risk factors for TB:**
- **Immunocompromised** (HIV, DM, steroids, elderly)
- **Low socioeconomic status** (crowding, malnutrition)
- **Born/live in endemic area**
- **Substance abuse** (smoking, drugs, alcohol)

## Primary TB infection

- Airborne droplets inhaled into alveoli
- **Macrophages engulf** but cannot destroy → **bacteria replicates within** → macrophages clump together = **granuloma**
- 1–2w later = **cell-mediated response** → immune cells surround macrophages = **Ghon focus** (usually mid-zone of lungs)
- Ghon focus + lymph node involvement = **1° complex**

**POSSIBLE OUTCOMES:**

→ Usually infection is cleared completely **with no / very mild symptoms only**
→ 5% get **full-blown symptoms** ± **dissemination** (see below)
→ Sometimes bacteria remain **dormant** within macrophages

## Secondary TB infection

- **Reactivation** of infection when **immunocompromised** (e.g. elderly, HIV)
- Bacteria replicated & spread to **upper lobes**
- Cytokine release causes necrosis & **cavitations in lung tissue**

**DISSEMINATION:** mycobacteria enter blood & spread throughout body
→ deposition of TB particles through lungs & other sites = **miliary TB**

| Site | Condition | Symptoms |
|---|---|---|
| **Brain** | Meningitis | Headache, seizures, meningism |
| **Kidneys** | Sterile pyuria | Dysuria, haematuria, frequency |
| **GI tract** | Ileocaecal/peritoneal | Abdo pain/mass, distension, ascites |
| **Vertebrae/joints** | Pott's disease / arthritis | Chronic back pain, painful joints |
| **Adrenal glands** | Addison's | Weight loss, thirst, fatigue, weakness |
| **Heart** | Pericarditis & effusion | Heart failure, arrhythmias, chest pain |
| **Lymph nodes** | Lymphadenitis | Enlarged nodes but painless |
| **Lung pleura** | Pleuritis | Painful breathing |

## Clinical features of pulmonary TB

1. **Early/non-specific:** fever, **drenching night sweats**, malaise/weakness/fatigue, **weight loss, lymphadenopathy**
2. **Late/specific:** productive cough (**sputum** & **haemoptysis**), pleuritic chest pain
3. **Signs of complications:** pneumonia, pleural effusion, empyema, haemoptysis

NB: pulmonary TB may be asymptomatic

## Investigations

**FOR ACTIVE TB**

Culture & microscopy = gold standard but takes 6w

1. **Bloods:** FBC, U&Es, LFT, CRP
2. **CXR** – upper lobe cavitation, pleural effusion, miliary shadowing
3. **Sputum culture:** 3× samples → Ziehl–Neelsen stain = acid-fast bacilli
4. **Bronchoscopy & alveolar lavage:** if sputum = –ve

**SCREEN FOR LATENT TB**

1. **Mantoux test:** tuberculin injection → redness >15mm = +ve
2. **Interferon gamma assay release:** shows previous exposure
3. **Consider HIV test**

## Management[4]

1. **Active:** R,I,P,E for 2m + R,I for 4m
2. **Latent:** R,I for 3m **or** I for 6m

compliance = key to successful Mx

**R** = rifampicin
**I** = isoniazid
**P** = pyrazinamide
**E** = ethambutol

| | Important side-effects |
|---|---|
| Rifampicin | Hepatitis, red/orange urine & tears, enzyme induction (OCP) |
| Isoniazid | Hepatitis, neuropathy, enzyme induction (OCP)* |
| Pyrazinamide | Hepatitis, arthralgia |
| Ethambutol | Optic neuritis – starts with loss of colour vision |

*OCP = oral contraceptive pill → affected by these medications

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[4] NICE CKS (2019) *Tuberculosis*

# Covid-19

**= an acute respiratory disease caused by a novel coronavirus**
→ clinical severity ranges from mild cold-like illness to severe pneumonia & acute respiratory distress

## Symptoms

### CHARACTERISTIC SYMPTOMS

- Fever
- Cough
- Dyspnoea

### OTHER COMMON SYMPTOMS

- Headache
- Sore throat
- Nasal congestion
- Fatigue, arthralgia, myalgia
- GI upset
- Red, dry, itchy eyes
- Change in taste/smell

### UNCOMMON SYMPTOMS

- Chest pain
- Haemoptysis
- Crackles/rales
- Oral mucosa lesions
- Tinnitus / hearing loss

### SEVERE DISEASE

- Tachypnoea, tachycardia
- Respiratory distress
- $SpO_2$ <90% on air

± sepsis, shock, ARDS, acute thrombosis

## Investigations

- Antigen / lateral flow test – variable sensitivity
- Real-time PCR (RT–PCR) – confirms diagnosis
- Pulse oximetry – hypoxia can develop without obvious symptoms

**In patients with severe illness / respiratory distress:**

- ABG
- FBC – lymphopenia, leucocytosis, thrombocytopenia, ↓ eosinophils
- CRP, LFTs, U&Es, TFTs, blood glucose
- Coagulation screen – ↑ D-dimer, ↑ PT, ↑ fibrinogen, ↑ INR
- Cardiac biomarkers (CK-MB, BNP, troponin) – may be elevated in severe disease
- Blood & sputum cultures – to rule out sepsis & other causes of LRTI
- CXR – ground glass opacities, consolidation (usually bilateral and basal)
- CT chest – only if severe clinical Covid & CXR normal or unclear

## Management[5]

**MILD DISEASE:** isolation, infection control measures, symptom management, supportive care (encourage fluids)

**MODERATE–SEVERE DISEASE:** consider admission

- Oxygen therapy – if $SpO_2$ <90%
- Cautious IV fluids
- Venous thromboprophylaxis – if need oxygen & no increased bleeding risk – continue for minimum 7d
- Low dose haloperidol if delirium or agitation
- Consider empirical ABX – only if suspicion of secondary bacterial infection
- Steroids – in all with oxygen requirement (10d of 6mg PO dexamethasone OD or prednisolone in pregnancy)
- Consider IL-6 inhibitor – in moderate–severe disease + $O_2$ requirement + CRP >75, despite steroids

**CRITICAL DISEASE:** transfer to ICU or respiratory support unit

- Consider high flow nasal oxygen or non-invasive ventilation
- Consider invasive ventilation – if acutely deteriorating despite non-invasive ventilation
- **Consider extracorporeal membrane oxygenation (ECMO)** – if above methods fail
- **Consider monoclonal antibody therapy** – type given depends on strain of Covid (variant of concern test needed)

**PROPHYLAXIS FOR IMMUNOCOMPROMISED:** immediate treatment with MAB or antiviral as soon as they test positive

*Be aware of atypical presentations in the elderly*

**Differential diagnosis:**

- Community-acquired pneumonia
- Influenza / common cold
- Febrile neutropenia
- Other respiratory virus
- Pulmonary TB

**When is PCR testing advised?**

- new continuous cough / high temperature / altered smell or taste
- acute respiratory infection
- worsening of underlying respiratory illness

**Patients with negative PCR but still high clinical suspicion of Covid should be retested**

**Complications of Covid-19:**

- VTE
- CVS complications
- Liver injury
- AKI
- Neuro complications
- Long Covid

**Long Covid usually occurs 3m after infection & lasts ≥2m with no alternative diagnosis**

**Sx:** persistent cough, fever, dyspnoea, weakness, fatigue, loss of smell/taste, dizziness, GI upset

**Ix:** bloods, CXR, CT chest (r/o other diagnosis)

**Mx:** rest, breathing exercises, graded exercise return, psychological support

**Respiratory follow-up:** depends on severity of illness

*May need repeat CXR, pulmonary function tests, echo*

**Poor prognostic factors:**

- male
- >65y
- obesity
- smoker
- diabetes
- CVD
- respiratory disease
- immunosuppressed

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[5] BMJ Best Practice (2021 + current expert opinion) *COVID-19*

# Asthma

**Triggers**
- allergens: pets, pollen, dust mites
- cold air
- emotion
- smoking
- viral infection
- pollution
- drugs: NSAIDs/BBs

**Diagnostic test results**
- **FEV$_1$:FVC** <70%
- **PEF** >20% variability
- **BDR:** FEV$_1$ ≥12% improvement or ≥200ml volume increase
- **FeNO** >40ppb

**Key elements of diagnosis**
1. **History** – variable symptoms, triggers, PHx or FHx of atopy
2. **Examination** – wheeze
3. **Spirometry** – bronchodilator reversibility
4. **Response to trial of Tx**

NO = produced in response to inflammation

| Severe attack | Life-threatening attack |
|---|---|
| • incomplete sentences<br>• accessory muscles<br>• hyperinflated chest<br>• pulsus paradoxus* | • exhaustion/confusion<br>• silent chest<br>• cyanosis |
| • **PEF 33–50% of best**<br>• RR ≥25<br>• HR ≥110 | • **PEF <33% of best**<br>• $spO_2$ <92%<br>• ↓HR & ↓BP |
| *↓SBP with inspiration | ABG: ↑$CO_2$, $O_2$ <8, low pH |

**Parasympathetic action:** ACh → $M_3$ receptors = bronchoconstriction & ↑mucus
**Sympathetic action:** adrenaline → $\beta_2$ receptors = bronchodilation & ↓mucus

Chronic, reversible increases in airway resistance due to **bronchospasm, inflammation** & **mucus production**

## Pathophysiology

Triggers activate mast cells to release spasmogens & chemotaxins:

1. **Early phase: bronchospasm** (**spasmogens:** histamine, PGs, leukotrienes)
2. **Late phase: inflammation** (**chemotaxins:** attract eosinophils/monocytes)

## Types

1. **Extrinsic:** type 1 hypersensitivity reaction (↑ IgE ± other atopies)
   - ↳ early onset/younger patients (may improve with age)
   - ↳ in adults = OCCUPATIONAL ASTHMA: chemicals, enzymes in flour, animal substances
2. **Intrinsic:** non-immune mechanisms (often no cause identified)
   - ↳ late onset/middle-aged patients

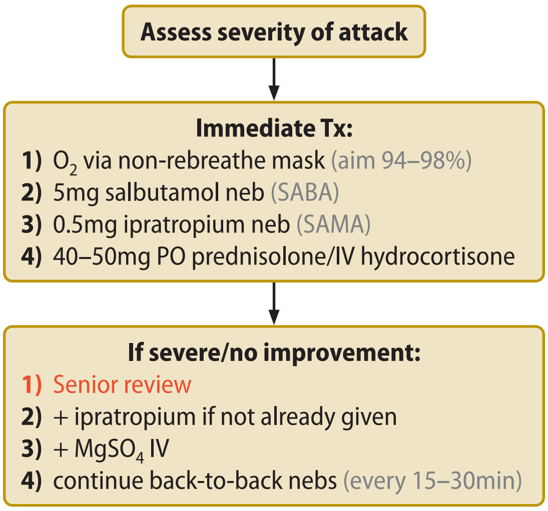
## Symptoms

- Wheeze, SOB, cough – worse at night / early morning / on exercise
- Chest tightness

## Investigations

- **Hx:** FHx/PHx of atopies, typical Sx with diurnal variation, identifiable trigger
- **Auscultation:** expiratory polyphonic wheeze
- **Atopy tests:** skin prick / serum IgE
- **Spirometry + bronchodilator reversibility test (BDR)**
- **FeNO test (fraction of expired NO)** – if still unsure of Dx in kids **or** if >17y
- **PEF:** monitor variability over 2–4w
- **Direct bronchial challenge:** last resort

## Management of acute asthma (adults)[6]



**Assess severity of attack**

**Immediate Tx:**
1) $O_2$ via non-rebreathe mask (aim 94–98%)
2) 5mg salbutamol neb (SABA)
3) 0.5mg ipratropium neb (SAMA)
4) 40–50mg PO prednisolone/IV hydrocortisone

**If severe/no improvement:**
1) Senior review
2) + ipratropium if not already given
3) + $MgSO_4$ IV
4) continue back-to-back nebs (every 15–30min)

**Fig. 9.4**

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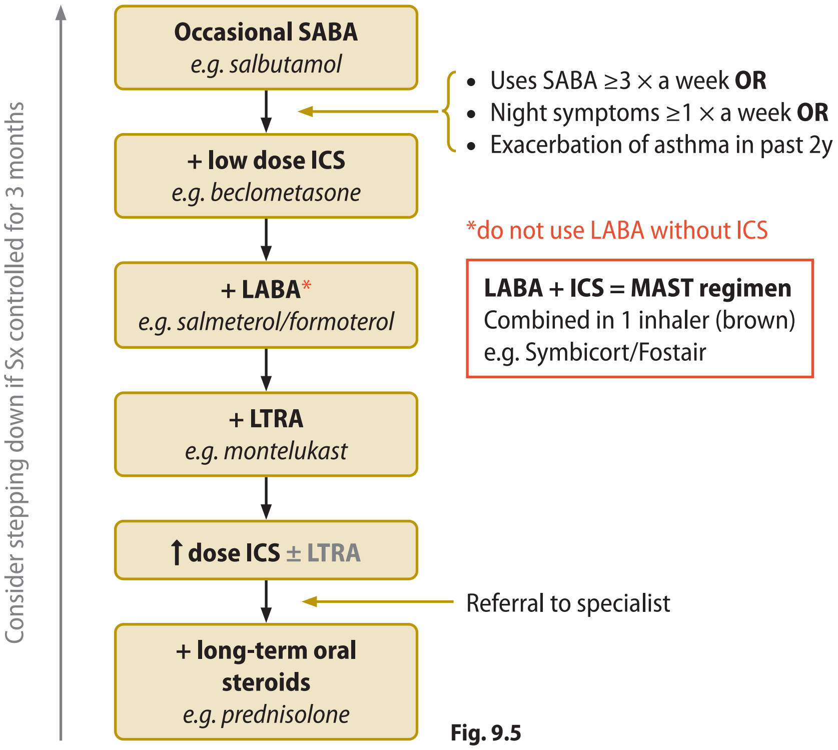
[6] NICE CKS – *Clinical scenario: Acute exacerbation of asthma*

## Management of chronic asthma[7]


Consider stepping down if Sx controlled for 3 months
Occasional SABA
e.g. salbutamol
+ low dose ICS
e.g. beclometasone
+ LABA*
e.g. salmeterol/formoterol
+ LTRA
e.g. montelukast
↑ dose ICS ± LTRA
+ long-term oral steroids
e.g. prednisolone
Uses SABA ≥3 × a week OR
Night symptoms ≥1 × a week OR
Exacerbation of asthma in past 2y
Referral to specialist

*do not use LABA without ICS

**LABA + ICS = MAST regimen**
Combined in 1 inhaler (brown)
e.g. Symbicort/Fostair

**Fig. 9.5**

### OTHER FACTORS TO CONSIDER:

1. Lifestyle: smoking, weight loss, breathing exercises
2. Inhaler technique: spacer, add-ons
3. Safety-net: including signs & Mx of acute attack
4. Follow-up appointment: annual review with practice nurse

## Safety-netting

### SEEK MEDICAL ATTENTION IF:

- Symptoms are getting worse/interfere with daily life
- Waking up at night

### SIGNS OF AN ACUTE ATTACK:

- Reliever inhaler isn't helping
- Too breathless to speak/eat/sleep
- Very tight chest / coughing a lot
- RR increasing / feel like can't get enough air

### Medications

1. **Beta-2 agonists (SABA/LABA)**
   - Beta-2 selective → relaxes smooth muscle in lungs

*SE = tachycardia, muscle cramps/tremors*

2. **Inhaled corticosteroids**
   - ↑ lipocortin → inhibits $PLA_2$
   - reduced arachidonic acid conversion to LTs/PGs
   - ↓ inflammation

*SE = oral candidiasis **(rinse mouth)***

3. **Leukotriene receptor antagonists (LTRAs)**
   - ↓ bronchoconstriction & mucus
   - ↓ eosinophils & inflammation

*SE = GI upset, headache, hepatic disorder, Churg–Strauss syndrome (vessel inflammation)*

4. **Long-term oral steroids**
   - same mechanism as ICS
   - more systemic SEs

*SE : acne, hair, ↑ weight, HTN, cataracts, osteoporosis, immunosuppression/infections*
→ cause adrenal suppression so ↓ dose gradually

### Components of an asthma review

- Level of control – poor control suggested if:
  - using SABA >3× per week
  - night symptoms
  - interfering with activities
  - chest tightness, wheeze
- Any exacerbations
- Compliance/technique
- Side-effects of medications

### What to do in an asthma attack

- Sit up straight
- Puff of PRN inhaler – up to 10 times
- If no improvement call 999

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[7] BTS/SIGN (2019) *Guideline for the management of asthma*

# Chronic obstructive pulmonary disease

Chronic, progressive, **poorly reversible** airway obstruction including **chronic bronchitis & emphysema**

**Risk factors for COPD:**

- Smoking
- Occupational dust
- Childhood infections
- α1 antitrypsin deficiency

Consider if young / no other risk factors

## Pathophysiology

**CHRONIC BRONCHITIS:** ↑ mucus production & inflammatory cells → scarred/ thickened epithelium → ↑ airway resistance

- Chronic productive cough (lasting ≥3m in 2 successive years)
- Poor alveolar ventilation = T2RF: ↓$O_2$ & ↑$CO_2$ (**lose hypoxic drive**)

**EMPHYSEMA:** ↑ protease activity destroys alveoli → ↓ elasticity & recoil, enlarged air spaces (↓ surface area)

- ↑ RR & HR (compensate for ↓ gas exchange)
- Cachexia (high energy demand for respiration)
- Poor gas exchange = T1RF: ↓$O_2$ & normal $CO_2$

**BRONCHIOLITIS / ACUTE BRONCHITIS:** short-term inflammation (usually viral)

## Symptoms

- Productive cough – clear, white sputum
- Progressive dyspnoea + wheeze
- Frequent LRTIs

## Signs

**Inspection:** ↑ RR, flapping tremor, cyanosis, barrel-chest, pursed lips
**Palpation/percussion:** reduced chest expansion, hyperresonance
**Auscultation:** polyphonic expiratory wheeze, ↓ breath sounds

## Investigations

1. **Hx & examination:** suspect if symptoms in those >35y with a risk factor
   → **Ask about:** weight loss, fatigue, chest pain, haemoptysis paroxysmal nocturnal dyspnoea, ankle swelling, ↓ exercise tolerance
2. **Spirometry + BDR:** assess severity with Gold criteria
   - **$FEV_1$** <80%
   - **$FEV_1$:FVC** <0.7
   - **BDR:** no improvement
   - **PEF:** little variation
3. **CXR:** hyperinflation, flat diaphragms, ↓ peripheral markings
4. **Other:**
   - **FBC:** Hb, PCV (↑ CRP if infection)
   - **ABG:** ↓ $O_2$ ± ↑ $CO_2$
   - **ECG/echo:** cor pulmonale / RV hypertrophy
   - **Sputum culture:** r/o infection or bronchiectasis

| MRC Dyspnoea Scale | |
|---|---|
| 1 | Only SOB on strenuous exercise |
| 2 | SOB if hurrying / walking up hill |
| 3 | SOB on flat |
| 4 | Stop for breath after 100m |
| 5 | SOB with dressing etc. |

| Gold criteria | |
|---|---|
| **Mild** | $FEV_1$ >80% |
| **Moderate** | $FEV_1$ 50–79% |
| **Severe** | $FEV_1$ 30–49% |
| **V. severe** | $FEV_1$ <30% |

**Interpreting spirometry**

| | Obstructive | Restrictive |
|---|---|---|
| **$FEV_1$** | <80% | <80% |
| **FVC** | Normal | <80% |
| **$FEV_1$:FVC** | <0.7 | Normal |
| **Examples** | Asthma, COPD, bronchiectasis, CF | ILD, NMD, scoliosis, pulmonary oedema, obesity |

## Management of COPD[8]

**1. SMOKING CESSATION:** only intervention that ↓s mortality

- Nicotine replacement: patches, gum
- Varenicline: nicotine receptor partial agonist
- Bupropion: noradrenaline/dopamine reuptake inhibitor
- E-cigarettes: lack long-term evidence but **90% safer**

**2. OPTIMISE WEIGHT:** dietitian referral

- **BMI too high:** exercise / healthy diet
- **BMI too low:** nutritional supplements

---

[8] BTS–NICE Guideline (2010) *COPD in over 16s* [CG101]

### 3. PULMONARY REHAB: 3× a week for 6w

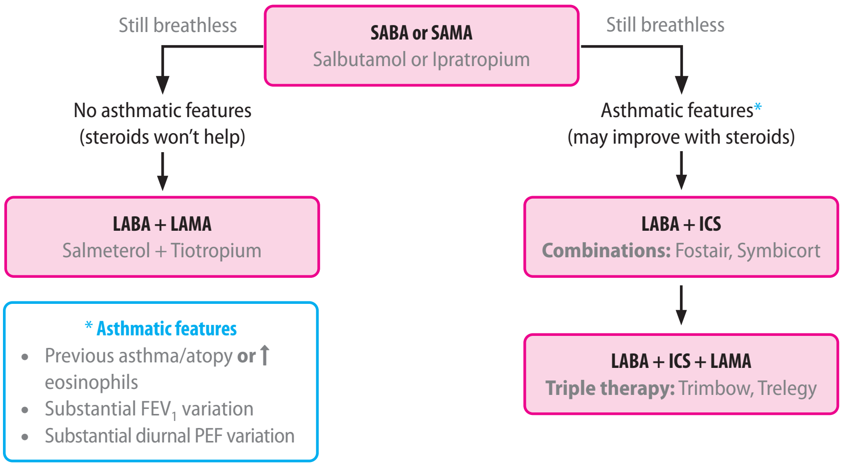
- Exercises to improve SOB
- Educate and promote self-management

### 4. MUCOLYTICS: e.g. carbocisteine

- May help chronic productive cough
- Review after 4w → stop if no benefit

**5. VACCINATIONS:** pneumococcal & flu vaccine

### 6. INHALED THERAPY: only if lifestyle interventions have been offered & still SOB


SABA or SAMA
Salbutamol or Ipratropium
Still breathless
Still breathless
No asthmatic features
(steroids won't help)
Asthmatic features*
(may improve with steroids)
LABA + LAMA
Salmeterol + Tiotropium
LABA + ICS
Combinations: Fostair, Symbicort
LABA + ICS + LAMA
Triple therapy: Trimbow, Trelegy
* Asthmatic features
Previous asthma/atopy or ↑ eosinophils
Substantial FEV1 variation
Substantial diurnal PEF variation

**** May consider theophylline after trialling SABA/LABA or if unsuitable for inhaled therapy****

**Fig. 9.6** Inhaled therapy.

## Management of acute COPD exacerbations

### SIGNS:

1. Altered sputum volume/colour
2. Reduced exercise tolerance
3. Fever/malaise/lethargy

### INVESTIGATIONS:

- Sputum sample if purulent
- ABG, FBC, U&Es
- CXR, ECG

### MANAGEMENT:

1. **Nebulised bronchodilators** (back-to-back): SABA & SAMA
   + theophylline if insufficient response
2. **Oral corticosteroids:** 30mg OD prednisolone for 5d
3. **Oxygen:** 88–92% via Venturi mask
4. **Antibiotics:** e.g. doxycycline
   If ≥2 of ↑ sputum, ↑ purulence, ↑ dyspnoea

## Annual follow-up

- Measure $FEV_1$ & FVC
- Assess function on MRC Dyspnoea Scale (see previous page)
- Review need for specialist referral

**ASTHMA–COPD OVERLAP SYNDROME (ACOS)**[9]
= COPD features of chronic airflow obstruction, combined with several features usually associated with asthma

**Symptoms:** persistent airflow limitation ($FEV_1$:FVC <0.7), but with some reversibility (better $FEV_1$ post bronchodilator)
**Investigations:** history, spirometry, CXR
**Management:** if diagnosis equally balanced between COPD & asthma, consider starting **ICS + LABA (&/or LAMA)**

**SABA/SAMA:** relieve acute bronchospasm
**LABA/LAMA:** ↑ $FEV_1$, TLC & improve SOB

**Antimuscarinic side-effects:**

- Constipation (can't poo)
- Urinary retention (can't wee)
- Dry mouth (can't speak)
- Blurred vision (can't see)
- Confusion (can't remember)

**Side-effects of other medications:**
Theophylline: headache, insomnia, tachycardia, N&V, arrhythmia, hypokalaemia
Varenicline/bupropion: GI upset, dry mouth taste disturbance, anxiety/depression, insomnia

**Safety-netting:** may give **rescue pack** (steroid + ABX)

**Warn of ↑ risk of pneumonia & signs of exacerbation**

- ↑ sputum volume/purulence
- ↓ exercise tolerance / ↑ SOB on exertion
- Worsening wheeze

**Warn of ↑ risk of pneumothorax**

- Sudden onset SOB
- Chest pain
- Cyanosis

**COPD complications:**

- **Acute exacerbations**
- Polycythaemia
- Respiratory failure
- Cor pulmonale
- Pneumothorax
- Lung carcinoma

---

[9] GOLD (2015) *Diagnosis of diseases of chronic airflow limitation: Asthma, COPD and ACOS*

# Bronchiectasis

Chronic inflammation of bronchi & bronchioles causing fibrosis & permanent dilation

## Causes/triggers of chronic inflammation

- **Congenital:** CF, primary ciliary dyskinesias (Kartagener's, Young's)
- **Infection:** measles, pertussis, pneumonia, TB, Covid
- **Bronchial obstruction:** COPD, foreign body, tumour
- **Connective tissue disease:** rheumatoid arthritis
- **Immune deficiency:** hypogammaglobulinaemia

## Symptoms

- **Persistent, productive cough:** lots of purulent sputum = foul-smelling
- **Intermittent haemoptysis**
- **SOB** – due to exacerbation
- **Recurrent febrile episodes** – recurrent exacerbations

Different to COPD:
much more sputum, haemoptysis, clubbing

## On examination

- **Clubbing**
- **Coarse inspiratory crackles**
- Wheeze if concurrent COPD/asthma
- Weight loss / thin
- **RVF:** ↑ JVP & peripheral oedema

## Investigations

1. **Bloods:** FBC, U&Es, CRP, clotting/coagulation screen
2. **Sputum sample:** MCS
3. **Spirometry:** obstructive pattern → irreversible (↓ $FEV_1$:FVC)
4. **CXR:** may show effusion, right ventricle hypertrophy, bronchial thickening
5. **HRCT:** assess distribution → **signet rings** = dilated airways
6. **Extra tests to determine cause:**
   - total Igs, specific antibodies, *Aspergillus* serology, CF sweat test & genetic screen
   - nasal nitrous oxide (NO) = low in primary ciliary dyskinesia (PCD)

## Management[10]

1. **Airway clearance:** chest physio (postural drainage) & mucolytics e.g. carbocisteine
2. **Promote smoking cessation** if appropriate
3. **Treat infection if present:** ABX guided by sputum culture
4. **Prophylactic antibiotics:** low dose azithromycin, nebulised anti-pseudomonal
5. **Annual flu vaccination**
6. **Bronchodilators:** if concurrent COPD/asthma, ABPA
7. **Corticosteroids:** if concurrent COPD/asthma, ABPA

+ assess for primary cause

**Complications of bronchiectasis:**
- Exacerbation/infection
- Haemoptysis
- Abscess/empyema
- Pneumothorax
- Right-sided heart failure
- Amyloidosis

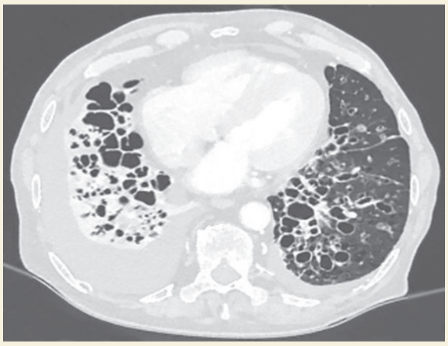


**Fig. 9.7** CT showing dilated airways.

**Organisms causing infective exacerbations:**
*H. influenzae, Strep. pneumoniae, Staph. aureus, Pseudomonas*

allergic bronchopulmonary aspergillosis

---

[10] BTS (2018) *Guideline for bronchiectasis in adults*

# Sleep apnoea[11]

→ ≥30 episodes of cessation of breathing, each lasting ≥10s over a period of 7h of sleep*

## Types

- **Obstructive:** due to upper airway collapse (↓ $O_2$ causes reflex of waking slightly & taking deep breath)
- **Central:** fault with central respiratory drive e.g. cerebral palsy, cognitive defect

## Symptoms

- Snoring/choking in sleep & witnessed apnoeas
- Restless/non-refreshing sleep
- Daytime sleepiness & ↓ concentration – **assess risk** e.g. driving
- Irritability & ↓ libido

**Kids:** poor school performance
**Babies:** poor feeding as blocked nasal breathing

## Investigations

- **History:** including fatigue levels
- **Examination:** upper airway endoscopy
- **Sleep studies:** measure pulse, ECG, $O_2$ overnight + audio/video recording of sleep*

→ **Polysomnography** = gold standard form of sleep study

## Management

1. **Lifestyle:** weight loss, smoking/alcohol
2. **Conservative:** nasal splints / tape & jaw advancers
3. **Medical:** CPAP via mask = noisy & uncomfortable BUT most tolerate
4. **Surgery**:** adenotonsillectomy, polypectomy, uvulopalatopharyngoplasty

**rarely indicated

1 & 2 also for management of simple snoring

**Risk factors:**

- **Older age**
- **Male**
- Down's
- Sedatives
- **Obesity**
- Smoking/alcohol
- Craniofacial abnormalities
- Neuromuscular disease

Epworth Sleepiness Scale objectively assesses levels of fatigue

*Sleep Apnoea Index: measures no. of episodes to determine severity

Must advise not to drive until symptoms are controlled

**Conditions associated with sleep apnoea:**

- HTN
- T2DM
- T2RF

---

[11] NICE (2021) *Obstructive sleep apnoea* (NG202)

# Cystic fibrosis

= an autosomal recessive condition affecting 1 in 2500 live Caucasian births (1 in 25 = carriers)

Consider CF in any child with recurrent chest infections, loose stools or failure to thrive

**Clinical manifestations**

| | |
|---|---|
| **Newborn** | Meconium ileus |
| **Infant** | Prolonged jaundice<br>Faltering growth<br>Recurrent chest infections<br>Malabsorption/steatorrhoea |
| **Young child** | Bronchiectasis<br>Nasal polyps / sinusitis<br>Rickets |
| **Older child/ adolescent** | ABPA<br>Diabetes mellitus<br>Premature osteopenia<br>Cirrhosis & portal hypertension<br>Renal/gallstones<br>Pneumothorax/haemoptysis<br>Male sterility<br>Rectal prolapse |

**Clinical signs on examination:**

- Lung hyperinflation
- Coarse inspiratory crepitations
- **Expiratory wheeze**
- Finger clubbing

Median survival = 47y

CFTR modulators are **gene-specific** but they are now available for 85–90% of adult cases → significant improvements in lung function & QoL

**Ix to assess for complications**

- Bloods: FBC, LFT, U&Es, CRP, clotting, amylase, glucose
- CXR
- Sputum cultures
- Spirometry = obstructive
- USS abdo: fatty liver, cirrhosis

## Pathogenesis

- Defective CF transmembrane conductance regulator (CFTR) **= abnormal $Cl^-$ transport across epithelial cells**
- Mutation on chromosome 7 **(ΔF508)** = most common
- Causes **thickened mucus secretions and ciliary dysfunction**

## Symptoms

**LUNGS:** impaired ciliary function & mucopurulent secretions

- **Recurrent chest infections:** *Staph. aureus, H. influenzae, Pseudomonas, Burkholderia* Hard to treat
  → Persistent cough with purulent sputum
  → **Complications:** bronchiectasis, abscess

**INTESTINE:** thick, viscous meconium

- **Meconium ileus (10–20%)** = intestinal obstruction
  → Vomiting & abdominal distension
  → Fail to pass meconium in first few days of life

**PANCREAS:** pancreatic duct blocked = enzyme insufficiency

- **Malabsorption & maldigestion**
  → Large, pale, offensive, greasy stools = steatorrhoea
  → **Complications:** failure to thrive / faltering growth

**SKIN:** abnormal sweat gland function

- **Excessive Na & Cl in sweat** (very salty)

## Diagnosis

- **Screening with heel prick:** ↑ immunoreactive trypsinogen (IRT)
- **Genetic screen for *CFTR* mutations:** if ↑ IRT
- **Sweat test:** if 2 *CFTR* mutations to confirm diagnosis → $Cl^-$ >60mmol/L

## Management

**RESPIRATORY:**

Mx involves MDT input

1. **Regular lung function review:** spirometry
2. **Chest physio:** 2× daily for 20–30min
   - **Younger:** parents perform percussion & positional drainage
   - **Older:** controlled deep breathing & physical exercise
3. **Prophylactic daily ABX:** + rescue stash for exacerbations
   - Daily flucloxacillin PO up to 3y, maybe 6y of age
   - Regular azithromycin (macrolide) neb
4. **Tx of persisting symptoms:** 14d IV ABX via PIC line
5. **DNAse / hypertonic saline:** reduce viscosity of secretions
6. **CFTR modulators:** partly correct defective CFTR protein

→ Bilateral lung transplant = only option for end-stage CF lung disease

**NUTRITION:**

1. **Regular dietary status assessment:** vitamins + high calorie diet (150% of normal)
2. **Pancreatic insufficiency Tx:** oral enzyme replacement tablets with all food (Creon)

**REGULAR MONITORING:** review at least once every 3m in a specialist centre

1. **Respiratory function**
2. **Nutritional status & growth monitoring**
3. **For complications:** DM, liver disease, bowel obstruction, pneumothorax, sterility etc.

# Lung cancer

**Primary lung cancer:** small cell or non-small cell
**Secondary lung cancer:** haematogenous & lymph spread from **kidneys, bones, breast, ovaries, prostate, GI tract**

**General RFs:** FHx, >50y, COPD/fibrosis, **smoking**

**3rd most common UK cancer**
72% cases due to smoking
**Peak age:** 85–89y

| Type | | Location | Growth | Prognosis | Complications | Risk factors |
|---|---|---|---|---|---|---|
| **Small cell 20%**<br>= neuroendocrine cells | | **Central**<br>(bronchi) | **Rapid**<br>Early mets | **Poor**<br>(months to years) | **Hormone secretion**<br>ADH = SIADH<br>ACTH = Cushing's<br>**Lambert–Eaton myasthenic syndrome**<br>prox. muscle weakness | **SMOKING**<br>**2nd hand smoke**<br>Radon gas<br>Asbestos |
| **Non-small cell 80%** | **Squamous cell 40%**<br>ciliated columnar | **Central**<br>(bronchi) | **Slow**<br>Local invasion<br>Late mets | **Better**<br>Curative surgery or radiotherapy + chemotherapy if present early & fit for Tx | **Bronchial obstruction**<br>Lung collapse<br>**Hypercalcaemia:** PTHrP | **SMOKING** |
| | **Adenocarcinoma 30%**<br>type 2 pneumocytes | **Peripheral** | **Slow**<br>LN invasion<br>Late mets | | **Pleural effusion**<br>SOB, pleurisy | **Asbestos**<br>often non-smokers |
| | **Large cell anaplastic 10%**<br>poorly differentiated epithelial cells | **Anywhere** | **Moderate**<br>LN invasion<br>Early mets | **Poor**<br>early mets = too late to operate | | Mix of all risk factors |

## Clinical presentation

- **Persistent cough:** >3m = **2ww referral**
- **Haemoptysis** in 70% if tumour ulcerates = need CXR
- **Dyspnoea / wheeze**
- **Chest pain** = late sign
- **Recurrent infection:** always recheck CXR 6w after pneumonia
- **General:** fever, night sweats, weight loss

## On examination

- Cachexia
- Lymphadenopathy
- Anaemia
- Chest signs – pleural effusion, lung collapse, consolidation
- Hypertrophic pulmonary osteoarthropathy = finger clubbing, wrist & ankle swelling

↳ not always all present

## Investigations

1. **Careful Hx & examination** – incl. FHx, occupation, smoking Hx
2. **Bloods:** FBC (anaemia), CRP (pneumonia), LFT (if jaundice), U&Es (SIADH), calcium (if confused)
3. **Imaging:** CXR → contrast-enhanced CT & PET if abnormal features on CXR
4. **Interventional radiology:** bronchoscopy & biopsy/EBUS
5. **Cytology:** of sputum & pleural effusion if present

EBUS – endobronchial ultrasound

## Management

depends on histology & stage → MDT

1. **Supportive/palliative**
→ **Pain & SOB:** opiates, anxiolytics, bronchodilators
→ **Nutritional support:** NG tube / PEG
→ **Obstructive Sx:** radiotherapy, stenting
2. **Small cell carcinoma: nearly always palliative** → radio-/chemotherapy
3. **Non-small cell cancer: surgical excision or curative radiotherapy**
→ if peripheral & no mets (chemo if advanced)

**Features of metastatic spread:**

- **Bone:** pathological fracture, anaemia, back pain
- **Brain:** CNS signs ataxia, seizures, confusion
- **Spinal cord:** compression Sx e.g. cauda equina
- **Liver:** hepatomegaly, jaundice
- **Heart/pericardium:** arrhythmia, pericarditis
- **Pancoast tumour:** Horner's, SVC obstruction
- **Recurrent laryngeal nerve:** hoarse voice

**Features of paraneoplastic syndromes:**

- **SIADH:** ↓Na, $H_20$ retention
- **Cushing's:** weight gain, acne, thin skin, HTN
- **Hypercalcaemia:** stones, bones, groans, moans

**Indications for 2ww referral**

**Patients >40y if:**

- ≥2 Sx of lung cancer **OR**
- 1 Sx & smoking Hx **OR**
- Any of:
  - persistent/recurrent chest infection
  - finger clubbing
  - persistent lymphadenopathy
  - chest signs consistent with lung cancer

# Sarcoidosis

Chronic granulomatous disorder that affects multiple organs including skin, lungs and eyes

## Aetiology

Thought to be a combination of genetic, environmental and infectious factors

## Symptoms

**Systemic:** fatigue, arthralgia, lymphadenopathy, low grade fever, weight loss
**Pulmonary:** cough, dyspnoea, wheezing
**Skin:** plaques, lupus pernio, erythema nodosum
**Eyes:** anterior uveitis, photophobia
**Cardiac:** heart block, cardiomyopathy
**Neurological:** headaches, seizures

## Investigations

1. **Bloods:**
    - FBC – leucopenia
    - U&Es – deranged if renal involvement
    - LFTs – may be deranged
    - Ca – raised
2. **24h urine calcium** – hypercalciuria
3. **CXR:** bilateral hilar lymphadenopathy ± pulmonary infiltrates ± fibrosis
4. **HRCT / CT TAP:** bilateral hilar lymphadenopathy, infiltrates
5. **Flexible bronchoscopy + endobronchial US (EBUS):** shows non-caseating granuloma, multinucleated giant cells
6. **Skin biopsies:** of any suspicious lesions
7. **Pulmonary function tests:** monitor disease progression
8. **ECG:** abnormal rhythm/conduction if cardiac involvement

## Management[12]

**Pulmonary disease:**

Annual flu and pneumococcal vaccines

- No treatment if asymptomatic (usually self-limiting)
- **1st line:** PO/inhaled corticosteroids
- **2nd line:** cytotoxics (methotrexate, azathioprine)
- **End-stage lung disease:** lung transplant

**Cutaneous disease:**

- Corticosteroids (topical → oral)
- Hydroxychloroquine

**Ocular disease:**

- Topical/PO corticosteroids

**Neurological disease:**

- PO corticosteroids

**Risk factors:**

- 20–40y
- FHx sarcoidosis
- Scandinavian

**Differentials:**

- TB
- histoplasmosis
- lung cancer
- lymphoma

**Monitoring:** 3–6m intervals

- FBC, LFTs, Ca, serum ACE levels
- Bone density screening every 2y

**Complications:**

- Cytotoxic-related side-effects
- Heart block, arrhythmias
- Neuropathy
- Glaucoma, cataracts
- Haemoptysis
- Infection

---

[12] BMJ Best Practice (2021) *Sarcoidosis*

# Interstitial lung disease

= general term describing conditions causing **fibrosis of interstitium**

## Pathogenesis

- Repeated exposure to exo-/endogenous stimuli = **inflammatory response**
- CK release, myofibroblasts & collagen deposition = **fibrotic foci**

## Clinical presentation

**Symptoms:**

- Dyspnoea on exertion – progressive
- Dry, non-productive cough

**Signs:**

- Fine inspiratory crackles
- ↓ Lung expansion
- Bronchial breath sounds
- Clubbing

## Investigations

1. **CXR & CT scan = gold standard**
   → Local fibrosis = 'streaky' shadows
   → Global fibrosis = 'honeycomb'/'ground glass'
2. **Lung function tests:** restrictive spirometry
3. **Blood tests:** for cause (e.g. RF) or avian precipitin
4. **Biopsy:** not routine → if unsure of Dx

## Causes

1. **Idiopathic pulmonary fibrosis:** genetic predisposition + environmental triggers
   ↳ RFs: >60y, male, smoking
2. **Endogenous stimuli:** CTDs (scleroderma), RA, sarcoidosis
3. **Exogenous stimuli:**
   - **Occupation:** asbestos, silicone, coal dust
   - **Drugs:** nitrofurantoin, amiodarone, MTX, SSRIs, radiotherapy
   - **Hypersensitivity reaction:** extrinsic allergic alveolitis
   - **Infections:** TB, fungal, viral, Covid-19
   - **GORD**

## Management[13]

**Confirm Dx & exclude treatable pathology / acute exacerbation** (e.g. active infection / hypersensitivity)

**General:**

1. **Lifestyle:** smoking cessation, avoid exposures
2. **Pulmonary rehab:** exercises to ↑ ability to cope with disease
3. **Monitoring:** assess if progressive or static
4. **$O_2$ therapy:** if persistent hypoxia **or** hypoxic on exertion (ambulatory $O_2$)
5. **Lung transplant:** curative (cut-off = 65y so most are too old)

**Specific therapies:**

1. **Idiopathic pulmonary fibrosis:** anti-fibrotics (nintedanib, pirfenidone)
   → only act to **slow disease progression**, do not improve disease
2. **Hypersensitivity pneumonitis:** steroids / steroid-sparing agents

**Differentials:**

- COPD
- Bronchiectasis
- Heart failure
- Lung malignancy

**Complications of fibrosis:**

- Type 1 respiratory failure
- Pulmonary HTN
- Lung cancer

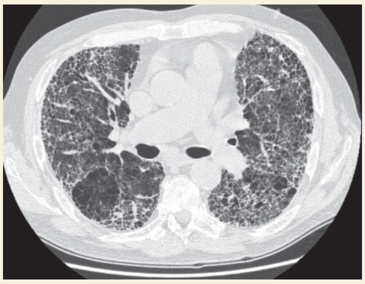


**Fig. 9.8** CT thorax of ILD.

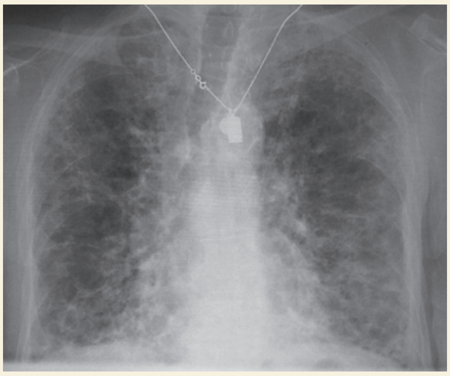


**Fig. 9.9** CXR of ILD.

**Key questions to ask:**

1. **Quantify SOB:** how far can you walk?
2. **Associated Sx:** cardiac, GI, infective
3. **PMHx:** GORD, sarcoidosis, RA, Sjögren's
4. **DHx:** nitrofurantoin, amiodarone, MTX
5. **SHx: smoking**, occupation, pets

**Mostly Tx is supportive (no cure)**
Consider palliative treatment in advanced disease

### EXTRINSIC ALLERGIC ALVEOLITIS / HYPERSENSITIVITY PNEUMONITIS (EEA/HP)

**Pathogenesis:** hypersensitivity reaction to inhaled organic antigen to which previous sensitisation has occurred

**Cause:**

- **Fungal spores:** *Aspergillus* = farmer's lung
- **Bird droppings:** avian protein = pigeon fancier's

**Diagnosis:**

- **Thorough Hx**
- Bloods: avian precipitant
- CXR & CT
- Lung function tests

**Management:**

- **Allergen avoidance**
- Steroids
- Steroid-sparing agents

**Acute Mx:** $O_2$ + PO prednisolone

Short intense exposure = reversible
Chronic exposure = irreversible

---

[13] NICE (2013, updated 2017) *Idiopathic pulmonary fibrosis* [CG163]

# Occupational lung disease

**Risk factors:** shipyard, plumbing, construction, carpentry
- Risk proportional to intensity of exposure
- May be up to 50y delay in onset

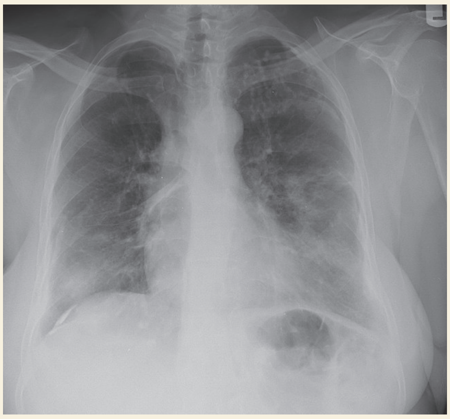


**Fig. 9.10** Pleural plaques.

**Causes of pneumoconiosis:** coal, asbestos, silica, cotton

## Asbestos-related

**PLEURAL PLAQUES** = benign (not pre-malignant)
- Following light exposure
- Asymptomatic and non-progressive
- **Mx:** reassurance

**PLEURAL THICKENING & EFFUSION** = benign (not pre-malignant)
- May cause SOB / restrictive spirometry
- Usually non-progressive
- **Mx:** reassurance → may get **compensation**

**ASBESTOSIS** = interstitial lung disease 2° to asbestos
- **Sx of ILD:** progressive SOB, dry cough, clubbing, fine expiratory crackles
- **Mx:** as for idiopathic pulmonary fibrosis → **compensation**

**MESOTHELIOMA** = malignant tumour of pleura
- Only need minimal exposure Poor prognosis – 2y survival
- **Sx:** SOB/wheeze, pleuritic pain, pleural effusions, B symptoms
- **Mx:** drain effusions, radiotherapy, **palliative, compensation**

## Pneumoconiosis

*→ lung disease due to occupational exposure*

**SIMPLE COAL WORKERS' PNEUMOCONIOSIS (CWP)** = inhaled coal dust particles trigger inflammatory response & fibrosis
- **Sx:** often asymptomatic
- **CXR:** multiple round opacities (<10mm) in upper zones
- **Mx:** supportive if symptomatic ($O_2$)

**COMPLEX CWP (PROGRESSIVE MASSIVE FIBROSIS)** = if continued exposure
- **Sx:** progressive SOB, productive cough (may be black)
- **CXR:** round opacities (>1cm) in mid-upper zones
- **Mx:** supportive ($O_2$) → **compensation**

## Occupational asthma

*→ responsible for 1 in 10 of adult-onset asthma*

**Causes:** bakeries (flour), animal fur, farms, car repair sprays, wood dust, soldering materials, hair salons (bleach)
**History:** asthma = worse at work & improves when on holiday
**Diagnosis:** difference in PEF at work & on holiday
**Management:** alter work pattern / protective equipment

**Remembering causes of ILD:** '5 boxes'

1. **Unknown box** = idiopathic interstitial lung disease (most common)
2. **Toolbox** = occupational (silica, coal, asbestos)
3. **Nesting box** = pigeon fancier's lung (and farmer's lung)
4. **Drug box** = medications (amiodarone, MTX, nitrofurantoin, SSRIs – fluoxetine)
5. **Glove box** = CTDs (Raynaud's, dysphagia, dry eyes/mouth)

# GENERAL SURGICAL PRINCIPLES

# 10

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## ABBREVIATIONS

**AAA** – Abdominal aortic aneurysm
**ABPI** – Ankle brachial pressure index
**ABPM** – Ambulatory blood pressure monitoring
**ACEi** – Angiotensin-converting enzyme inhibitor
**ALND** – Axillary lymph node dissection
**ALP** – Alkaline phosphatase
**ANA** – Antinuclear antibody
**APS** – Antiphospholipid syndrome
**APTT** – Activated partial thromboplastin time
**ARB** – Angiotensin receptor blocker
**ARDS** – Acute respiratory distress syndrome
**ASIS** – Anterior superior iliac spine
**AST** – Aspartate aminotransferase
**BB** – Beta-blocker
**CEA** – Carcinoembryonic antigen
**CF** – Cystic fibrosis
**CHF** – Congestive heart failure
**CLI** – Chronic limb ischaemia
**CLTI** – Critical limb-threatening ischaemia
**CMV** – Cytomegalovirus
**COCP** – Combined oral contraceptive pill
**CRT** – Capillary refill time
**CTD** – Connective tissue disease
**DHP** – Dihydropyridine
**DIC** – Disseminated intravascular coagulation
**DKA** – Diabetic ketoacidosis
**DM** – Diabetes mellitus
**DRE** – Digital rectal exam
**EAU** – Examination under anaesthetic
**EBV** – Epstein–Barr virus
**ER** – Oestrogen receptor
**ERCP** – Endoscopic retrograde cholangiopancreatography
**EVAR** – Endovascular aneurysm repair
**FAP** – Familial adenomatous polyposis
**FB** – Foreign body
**FNA(C)** – Fine needle aspiration (cytology)
**GA** – General anaesthetic
**GORD** – Gastro-oesophageal reflux disease
**GTN** – Glyceryl trinitrate
**HBPM** – Home blood pressure monitoring
**HDL** – High density lipoprotein
**HNPCC** – Hereditary non-polyposis colorectal cancer
**HRT** – Hormone replacement therapy
**HSV** – Herpes simplex virus
**IAP** – Intra-abdominal pressure
**IBD** – Inflammatory bowel disease
**IBS** – Irritable bowel syndrome
**ICA** – Internal carotid artery
**IDA** – Iron-deficiency anaemia
**IMA** – Inferior mesenteric artery
**IVC** – Inferior vena cava
**JVP** – Jugular venous pressure
**LDH** – Lactate dehydrogenase
**LDL** – Low density lipoprotein
**LIF** – Left iliac fossa
**LVH** – Left ventricular hypertrophy
**MRCP** – Magnetic resonance cholangiopancreatography
**NBM** – Nil by mouth
**OCP** – Oral contraceptive pill
**OGD** – Oesophago-gastroduodenoscopy
**PAD** – Peripheral arterial disease
**PCKD** – Polycystic kidney disease
**PET** – Positron emission tomography
**PID** – Pelvic inflammatory disease
**PR** – Per rectum
**PSA** – Prostate-specific antigen
**PT** – Prothrombin time
**PV** – Per vagina
**RAAS** – Renin–angiotensin–aldosterone system
**RIF** – Right iliac fossa
**RV** – Right ventricle
**SB** – Small bowel
**SCA** – Subclavian artery
**SCV** – Subclavian vein
**SLE** – Systemic lupus erythematosus
**SLNB** – Sentinel lymph node biopsy
**SMA** – Superior mesenteric artery
**SMV** – Superior mesenteric vein
**SV** – Stroke volume
**TB** – Tuberculosis
**UC** – Ulcerative colitis
**UO** – Urine output
**VTE** – Venous thromboembolism

# Wound healing

## Stages of healing

- **Acute wound:** follows normal, expected rate of healing e.g. surgical wound / simple laceration / burn
- **Chronic wound:** does not heal in orderly stages / expected time e.g. pressure / diabetic ulcer, fungating wound

| **1. Haemostasis** (within mins) | **2. Inflammatory** (usually settles by 3d) | **3. Proliferative** (up to 1m) | **4. Remodelling** (months to years) |
|---|---|---|---|
| **Prevents excess blood loss**<br>1. Platelets form **clot**<br>2. Vasoconstriction **(pallor)** | 1. Vasodilation = infiltration of cells & **oedema**<br>2. WBCs remove orgs/debris<br>3. ↑ blood flow = **erythema, warmth & swelling** | 1. Growth factors stimulate **angiogenesis** & $O_2$/nutrient delivery<br>2. Fibroblasts produce **collagen**<br>3. **Granulation tissue = moist & red**<br>4. Wound edge contraction<br>5. **Epithelial cells migrate** across wound | 1. **Strengthening & organisation** of collagen<br>2. Reduced vascularisation<br>3. **Scar tissue = WHITE** |

- **Healing by primary intention:** clean wound, no tissue loss, edges can be closely apposed (no scarring)
- **Healing by secondary intention:** tissue loss, or complication (e.g. infection) prevents close apposition of edges (granulation tissue and scarring)

## Wound infection

**Signs:** 5–7d post-op

1. **Local infection:** bleeding, red/swollen, change in pain / wound size / exudate
2. **Spreading infection:** spreading redness, swelling & inflammation, malaise, anorexia
3. **Systemic infection (sepsis):** fever, tachycardia, tachypnoea, confusion, shock

**Investigations:**

1. **FBC:** WCC, CRP
2. **Wound swab:** for MCS* → diagnostic & guides ABX treatment

*also check any microbiology available from the time of surgery

### 1. NECROTISING FASCIITIS: *Streptococcus pyogenes* or polymicrobial (anaerobes + aerobes)

→ **rapidly progressive infection** through subcutaneous tissue

→ 40% mortality = **surgical emergency**

**Symptoms:** pain, skin erythema/discolouration, skin ischaemia/necrosis → skin crepitus, vesicles/bullae

**Investigations:** bloods, blood cultures, VBG → imaging is not routine

**Management:** resuscitation & broad-spectrum ABX → **urgent surgical debridement** (± reconstructive surgery)

LRINEC = Laboratory Risk Indicator for Necrotising Fasciitis (based on CRP, WCC, Hb, sodium, glucose, creatinine)

### 2. ANAEROBIC (GAS) GANGRENE: *Clostridium perfringens*

→ infection of **muscle tissue** after injury/surgery resulting in **deep or contaminated wounds**

### 3. SYNERGISTIC (MELENEY'S) GANGRENE: *Streptococcus* + other organisms

→ spreading infection of **skin & subcutaneous tissues** starting from retention **sutures / other foreign** bodies postoperation

## Classification of surgical procedures & wound infection risk

| Category | Description | Infection risk |
|---|---|---|
| **Clean** | Incise uninfected skin & not viscera | <2% |
| **Clean-contaminated** | Breach of viscera (not colon) | 8–10% |
| **Contaminated** | Breach & spill viscera OR breach colon | 12–20% |
| **Dirty** | Site contaminated with pus or faeces | 25% |

### Dehisced (reopened) wounds

**Causes:** infection, haematoma, underlying fluid collection

**RFs:** age, DM/obesity/smoking, op >6h, post-op hypotension, excess coughing

**Mx:** wound cleansing & packing, –ve pressure therapy, tissue viability nurse (may need return to theatre)

### Factors affecting healing:

- Location & severity of injury
- Age, nutrition, comorbidities
- Infection of wound
- Smoking/alcohol

### Risk factors for wound infection:

- extremes of age
- DM/immunosuppression
- smoking, malnutrition
- poor wound closure
- surgery >2h
- CKD

Fournier's gangrene = necrotising fasciitis of the perineum

### Common infections post-op:

*Staphylococcus, Streptococcus, Pseudomonas*

# Postoperative care

## Summary of postoperative problems

| Immediate | Delayed |
|---|---|
| • Primary haemorrhage<br>• Shock<br>• Low urine output<br>• Pain<br>• N&V<br>• Confusion/delirium | • Infection (pneumonia, UTI, wound)<br>• Pressure sores<br>• Paralytic ileus<br>• Secondary haemorrhage (7–10d post)<br>• Acute MI (in first 72h)<br>**Immobilisation:**<br>• DVT, PE, stroke – give LMWH/aspirin<br>• Urinary retention & AKI<br>• Chest infections – in elderly/ventilated<br>• Atelectasis<br>• Pressure sores & muscle wasting |

### Common sources of post-op pyrexia & infection

**C**hest (infection)
**C**atheter (UTI)
**C**ut (infection)
**C**annula (infection)
**C**alves (DVT)
**C**entral line (infection)
**C**ollections (abdo/pelvis)

**Start SEPSIS 6 if potential sepsis** (qSOFA score ≥2 / clinical judgement)

**1–3d:** respiratory
**3–5d:** urinary
**5–7d:** wound/abscess/leak

**Ix:** blood cultures, CXR, urine dip / MCS, cannula site, surgical wound swab, CT/USS of surgical site

## Anastomotic leak

**AETIOLOGY:** **leak of luminal contents from a surgical join** → important complication of **GI surgery**

**RISK FACTORS:**

| Patient risk factors | Surgical risk factors |
|---|---|
| • Medication (steroids/ immunosuppressants)<br>• Smoking/alcohol<br>• DM, obesity, malnutrition | • **Emergency surgery**<br>• **Longer intra-operative time**<br>• Oesophageal–gastric or rectal anastomosis<br>• Peritoneal contamination (by free pus or faeces) |

**CLINICAL PRESENTATION: 5–7d post-op** (often due to ischaemia)

- Abdo pain ± peritonism
- **Fever**, tachycardia, new atrial fibrillation

→ consider in any patient **failing to progress** post GI resection

**INVESTIGATIONS:**

- FBC, CRP, U&Es, LFT, clotting, VBG
- Group & save
- **CT with contrast = diagnostic**

**MANAGEMENT:**

- **SEPSIS 6** (IV ABX)
- Larger leaks may need drainage or laparoscopic exploration / surgical intervention

## Intra-abdominal abscess

**RISK FACTORS:**

- Intra-abdominal infection (e.g. appendicitis, diverticulitis)
- Recent intra-abdominal surgery

**CLINICAL PRESENTATION:**

CT abdo with contrast
= diagnostic

- Fever
- Anorexia/N&V
- Abdominal pain
- Altered bowel habit / prolonged ileus

**MANAGEMENT:**

- IV ABX + drainage (+ send fluid for culture)
- May need to return to theatre

## Paralytic ileus

**AETIOLOGY: reduced intestinal motility** → very common following abdo/pelvic surgery

**RISK FACTORS:**

| Patient risk factors | Surgical risk factors |
|---|---|
| • Increased **age**<br>• **Electrolyte derangement** (e.g. Na$^+$, K$^+$ and Ca$^{2+}$)<br>• Neurological disorders (e.g. dementia/Parkinson's) | • Use of **anti-cholinergic/opioid** medication<br>• **Pelvic/abdominal surgery**<br>• Extensive **intra-operative intestinal handling**<br>• Peritoneal contamination (by free pus or faeces) |

**CLINICAL PRESENTATION:**

- Failure to pass faeces/flatus
- Abdo distension & **absent bowel sounds**
- N&V

*Ix are done to rule out more serious pathologies*

**MANAGEMENT:**

- IV fluids & daily bloods (electrolytes)
- Encourage mobilisation
- **Review analgesia (opiates)** in conjunction with pain team
- Consider if need to be NBM ± NG tube (remove stomach contents to reduce vomiting)

**INVESTIGATIONS:**

- FBC, CRP, U&Es
- Electrolytes (Ca$^{2+}$, PO$_4^-$, Mg$^{2+}$)
- **Consider imaging:** AXR ± CT (to rule out other pathology e.g. obstruction)

## Post-op haemorrhage

**Primary bleed** – within intra-operative period → resolved during operation & close monitoring post-op

**Reactive bleed** – within 24h of operation → usually a missed vessel / slipped ligature

**Secondary bleed** – 7–10d post-op → usually erosion of a vessel 2° to infection

*More detail on haemorrhagic shock in Chapter 16: Critical illness*

**SIGNS/SYMPTOMS:** tachycardia, tachypnoea, dizziness, reduced urine output (NB: hypotension = **late sign**)

**INVESTIGATIONS:** thorough examination for signs of bleeding/swelling/discolouration/tenderness/peritonism

**MANAGEMENT:**

1. **ABCDE** → IV fluid resuscitation + direct pressure on bleeding site if visible
2. **Urgent senior review** → may need to return to theatre
3. **Urgent blood transfusion** – if moderate/severe bleeding (activate major haemorrhage protocol if necessary)

## Urinary retention

**Risk factors for urinary retention:**

- >50y
- Spinal/epidural
- Neuro comorbidity
- Paralytic ileus
- Pelvic/uro surgery
- Opiates, antimuscarinics
- Infection/sepsis
- Constipation

**PRESENTATION:**

- Reduced output
- Suprapubic mass/pain

**INVESTIGATIONS:**

- USS bladder (residual volume)
- Kidney function: eGFR, U&Es

**MANAGEMENT:**

- Withdraw causative agents
- **Catheter**

**Work-up for AKI:**

- Fluid status
- FBC, CRP, U&Es, LFT
- Urine dip
- USS KUB

# Postoperative assessment

Use mnemonic: 'IMPORTANCE'

| | | |
|---|---|---|
| I | Introduction | Background of patient<br>(demographics, type of surgery & anaesthetic, any complications, days post-op) |
| M | Mental state | AVPU/AMT10 (compare to their baseline) |
| P | Pain | 1. **Identify:** SOCRATES<br>2. **Manage:** regular or PRN analgesia (check if any SEs & if it can it be ↑ or ↓) |
| O | Observations | 1. Calculate **EWS** & assess obs from last 24h<br>2. CVS/respiratory/abdominal exam if needed |
| R | Renal | 1. Assess **fluid chart** (urine output & fluid intake)<br>2. Check any **catheters** / surgical drains → assess output volume & colour |
| T | Thromboprophylaxis | 1. Check appropriate prophylaxis in place (LMWH, stockings, foot pumps)<br>2. Check calves for DVT, ask about SOB<br>3. **Mobilise ASAP** (encouragement, physio/OT) |
| A | Abdominal | 1. Are they eating/drinking?<br>2. Any N&V? Abdo pain?<br>3. Have they opened their bowels yet? |
| N | Neurovascular status | Check pulses & sensation → document clearly |
| C | Cut | Check post-op wounds, dressings & any drains |
| E | Exercise | Have they mobilised yet? (how far? any aids? does physio need contacting?) |

**Assessment of pain with 'SOCRATES'**

**S:** Site
**O:** Onset
**C:** Characteristic
**R:** Radiation
**A:** Associations
**T:** Timing
**E:** Exacerbating factors
**S:** Severity

## Other things to consider

- Any post-op bloods/imaging that need doing
- Discharge planning & follow-up arrangements
- Anyone else you need to contact (e.g. seniors, nurses, physio/OT, family)

# Fluid therapy

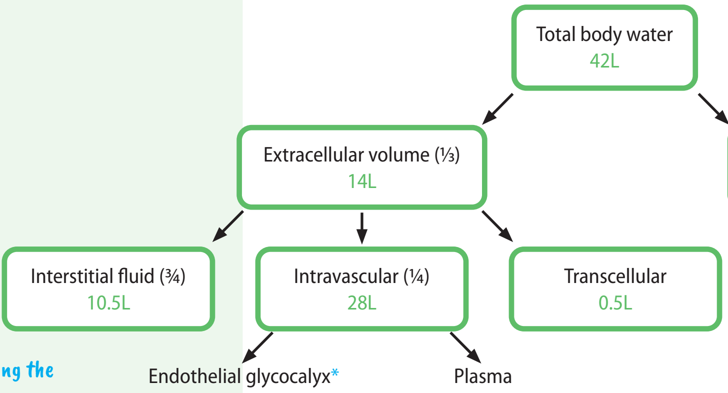
## Distribution of fluids

→ *fluid distribution is determined by extracellular $Na^+$ concentration*

- **Water:** distributed throughout total body water
- **Isotonic crystalloid:** stays in extracellular fluid → 0.9% NaCl
- **Hypertonic crystalloid:** draw fluid out of cells → 3% NaCl
- **Colloid:** intravascular (& some extracellular fluid)
- **Blood:** intravascular
- **Glucose 5%:** will distribute throughout all compartments (same as $H_2O$)

**Within extracellular fluid:**
75% goes to interstitial,
25% to intravascular

If a patient loses 1000ml of blood, 4000ml of 0.9% NaCl is needed (as 25% of this will replace the intravascular blood loss)



**Total body water** = 0.6 × weight
e.g. 70kg man has 42L TBW

*carbohydrate-rich layer lining the vascular endothelium

**Fig. 10.1**

## Assessing fluid status

- **History:** limits to intake, thirst, quantity & composition of losses
- **Examination:** pulse, BP (± orthostatic), CRT, JVP, oedema
- **Obs:** NEWS2, fluid balance charts / urine output, weight
- **Bloods:** U&Es, Cr, FBC
- **Medication review:** diuretics, antihypertensives

→ If still unsure of fluid status, can do **passive leg raise manoeuvre** (mimics increased fluid to heart) – **if >10% ↑ in SV** suggests patient will respond to fluid administration (need invasive monitoring of HR, BP & SV)

**Things to consider before prescribing fluids:**

1. Type of fluid loss
2. Concomitant electrolyte abnormalities
3. Renal & cardiac function

## Types of fluids

| | $Na^+$ (mmol/L) | $Cl^-$ (mmol/L) | $K^+$ (mmol/L) | Additives |
|---|---|---|---|---|
| **Plasma (EC fluid)** | **135–145** | **100–110** | **3.5–5.3** | **Lactate, calcium, glucose** |
| **0.9% NaCl** | 154 | 154* | | – |
| **Hartmann's** | 131 | 111 | 5 | Lactate, calcium |
| **5% dextrose** | – | – | | 50g/ml glucose |
| **0.18% NaCl + 4% dextrose** | 31 | 31 | | 40g/ml glucose |

* Risk of hyperchloraemic acidosis

**Colloids:** blood, dextrans, gelatines, human albumin solution
→ costly & risk of anaphylactoid reactions so rarely used

## Possible side-effects of fluid therapy

- Sodium overload (oedema)
- Hyperchloraemic acidosis
- Coagulopathy
- Fluid overload (pulmonary oedema, dilutional hyponatraemia)
  - → Stop IV fluids
  - → Furosemide
  - → SL/IV nitrate
  - → CPAP

## Principles of fluid therapy

1. Do not prescribe fluids pre-emptively → **regularly review & reassess to adjust fluids**
2. **Regular monitoring:** UO, BP, pulse, clinical signs of hypo-/hypervolaemia, U&Es
3. Clear prescription & fully completed fluid charts
4. Daily weights if have CCF
5. Switch from IV to enteral fluids ASAP

### Fluid checks

1. **Drug:** appropriate fluid chosen?
2. **Dose:** appropriate volume & rate calculated?
3. **Duration:** start & review/stop date documented?

## Resuscitation fluids

**Appropriate fluid:** 0.9% NaCl or Hartmann's

1. **250–500ml bolus over 15min**
2. **Reassess** (BP, UO, CRT, HR, RR)
3. Further bolus if needed (if >2L get help)

→ **Replace blood loss with:** RBC, FFP, platelet infusion

See *Chapter 16: Critical illness* for full table on hypovolaemic shock classification

**Beware:** do not blindly administer fluids if no idea of vital observations / type of shock

(IV fluids may exacerbate cardiogenic/obstructive shock)

**Classification of hypovolaemic shock:**

| Volume lost | Grade | Clinical signs |
|---|---|---|
| 15% (750ml) | 1 | Mild tachycardia, CRT 2–3sec |
| 15–30% (750–1500ml) | 2 | Tachycardia, cool peripheries, CRT 3–5sec |
| 30–40% (1500–2000ml) | 3 | Marked tachycardia & tachypnoea, **reduced BP, oliguria, confusion** |
| 40–50% (>2000ml) | 4 | Marked tachycardia, **low GCS, low BP** |

## Routine maintenance

**Appropriate fluid:** NaCl (0.18% or 0.9%) + dextrose (add $K^+$ if needed)

- Calculate daily requirements based on weight

e.g. 70kg patient needs 2100ml/d water (30ml × 70)

e.g. 70kg patient needs 70mmol/d $Na^+$, $Cl^-$, $K^+$ (approx. ½ bag 0.9% saline – as this contains 154mmol $Na^+$ & $Cl^-$)

**Cannot add $K^+$ to Hartmann's** so NaCl may be better choice of maintenance/replacement if need $K^+$ replacement

**Daily requirements account for:**

→ **1.5L** urine output/day → aim for **minimum 0.5ml/kg/h**

→ **500–800ml** insensible losses/day → increases if febrile

Fluid need increases 10% for every degree of pyrexia

**Additional sources of fluid loss to consider & account for:**

→ **GI losses:** if severe diarrhoea/vomiting or high stoma output

→ **Drains:** biliary, pleural, peritoneal

→ **Bleeding/burns**

**For potassium replacement either:**

+40mmol or +20mmol to saline

**Max peripheral infusion rate = 10mmol/h**

**Additional sources of electrolyte loss to consider & account for:**

→ **Sweating:** $Na^+$

→ **Diarrhoea / high stoma output:** $Na^+$, $K^+$ & $HCO_3^-$ (+ $Cl^-$ if stoma)

→ **Vomiting:** $K^+$, $Cl^-$, $H^+$ (hypochloraemic metabolic alkalosis)

**Daily requirements in health:**

- **Water:** 25–30ml/kg (1.5ml/kg/h)
- **Sodium:** 1mmol/kg
- **Potassium:** 1mmol/kg
- **Chloride:** 1mmol/kg
- **Glucose:** 50–100g

## Replacement

**Appropriate fluid:** NaCl (0.18% or 0.9%) + dextrose (aim for oral replacement if possible)

**Replacement = deficits + maintenance requirements**

1. Assess fluid status: examination, fluid balance chart, U&Es
2. Calculate maintenance requirements
3. Add in any deficits (previous & ongoing) e.g. D&V, sweating, stoma output
4. Consider 'complex' patients: old, obese, renal/liver/heart failure
5. **Monitor:** daily bloods & hydration status

*Appropriate rate of replacement: 1L over 4–6h*

**NB: in closed head injury** maintain euvolaemia with **2/3** of maintenance with **isotonic solution** to minimise **cerebral oedema**

# THE ACUTE ABDOMEN

# 11

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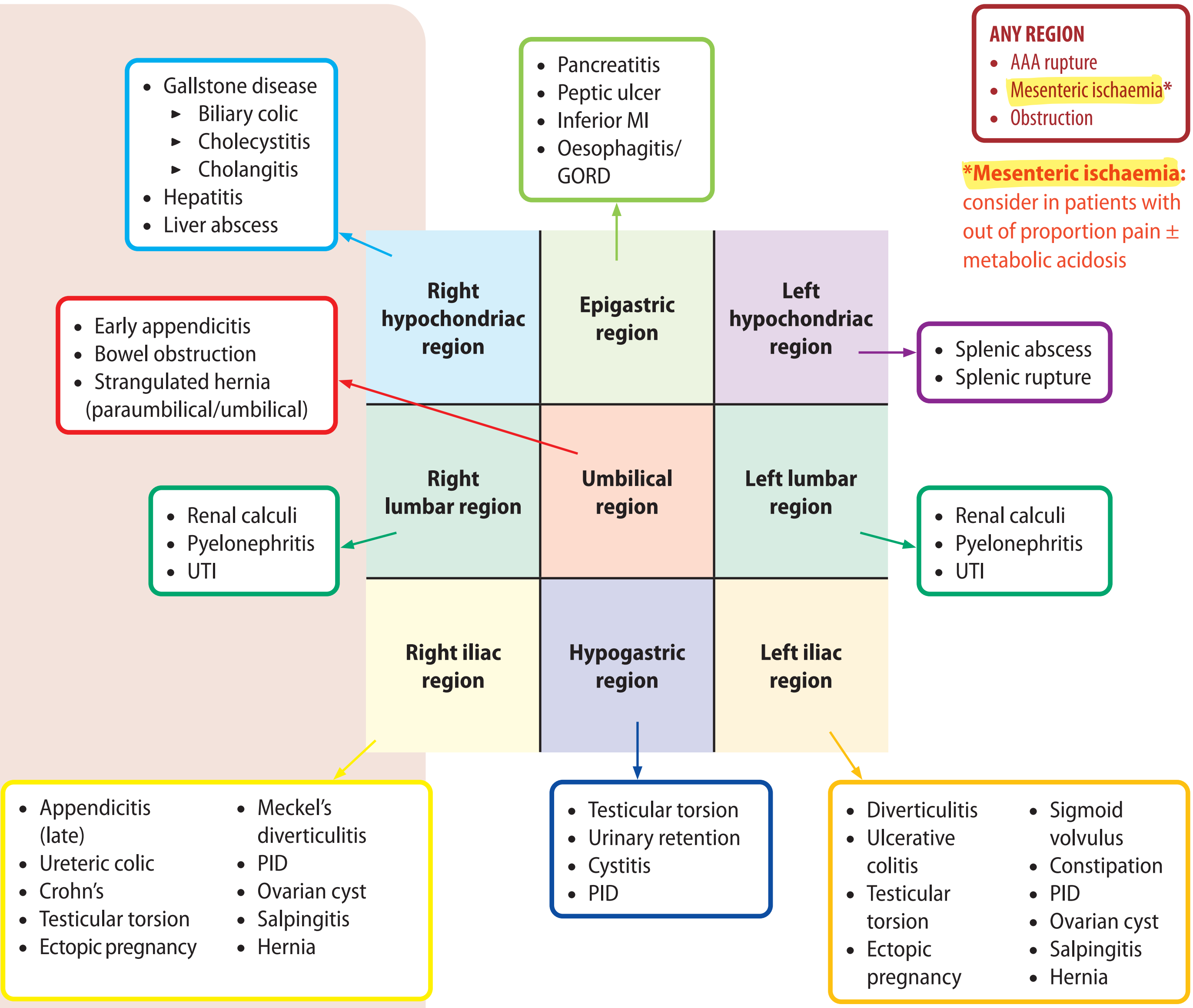
## ABBREVIATIONS

**AAA** – Abdominal aortic aneurysm
**ACEi** – Angiotensin-converting enzyme inhibitor
**ARDS** – Acute respiratory distress syndrome
**CF** – Cystic fibrosis
**CMV** – Cytomegalovirus
**CTAP** – Computed tomography abdomen & pelvis
**DIC** – Disseminated intravascular coagulation
**DKA** – Diabetic ketoacidosis
**DNACPR** – Do not attempt CPR
**DRE** – Digital rectal examination
**EBV** – Epstein–Barr virus
**ERCP** – Endoscopic retrograde cholangiopancreatography
**FB** – Foreign body
**GORD** – Gastro-oesophageal reflux disease
**IAP** – Intra-abdominal pressure
**IBD** – Inflammatory bowel disease
**IDA** – Iron-deficiency anaemia
**LHS** – Left-hand side
**LIF** – Left iliac fossa
**LN** – Lymph node
**MI** – Myocardial infarction
**MRCP** – Magnetic resonance cholangiopancreatography
**OGD** – Oesophagogastroduodenoscopy
**PID** – Pelvic inflammatory disease
**PR** – Per rectum
**PSHx** – Past surgical history
**RIF** – Right iliac fossa
**SLE** – Systemic lupus erythematosus
**SMA** – Superior mesenteric artery
**UC** – Ulcerative colitis
**UTI** – Urinary tract infection

# General overview



**ANY REGION**
- AAA rupture
- Mesenteric ischaemia*
- Obstruction

***Mesenteric ischaemia:** consider in patients with out of proportion pain ± metabolic acidosis

**Right hypochondriac region**
- Gallstone disease
  - Biliary colic
  - Cholecystitis
  - Cholangitis
- Hepatitis
- Liver abscess

**Epigastric region**
- Pancreatitis
- Peptic ulcer
- Inferior MI
- Oesophagitis/GORD

**Left hypochondriac region**
- Splenic abscess
- Splenic rupture

**Umbilical region**
- Early appendicitis
- Bowel obstruction
- Strangulated hernia (paraumbilical/umbilical)

**Right lumbar region**
- Renal calculi
- Pyelonephritis
- UTI

**Left lumbar region**
- Renal calculi
- Pyelonephritis
- UTI

**Right iliac region**
- Appendicitis (late)
- Ureteric colic
- Crohn's
- Testicular torsion
- Ectopic pregnancy
- Meckel's diverticulitis
- PID
- Ovarian cyst
- Salpingitis
- Hernia

**Hypogastric region**
- Testicular torsion
- Urinary retention
- Cystitis
- PID

**Left iliac region**
- Diverticulitis
- Ulcerative colitis
- Testicular torsion
- Ectopic pregnancy
- Sigmoid volvulus
- Constipation
- PID
- Ovarian cyst
- Salpingitis
- Hernia

**Fig. 11.1** This diagram should be used as a guide to the differentials of acute abdominal pain. It is important to note that any of the mentioned pathologies can present with pain in any area of the abdomen, and thus it is important to correlate location of pain with other clinical signs and investigations, and not to rule out differentials based on the location of pain alone.

## Medical causes of acute abdominal pain

- DKA
- UTI
- Basal lobe pneumonia
- Poisoning/overdose
- Addison's disease
- Hypercalcaemia
- Spontaneous bacterial peritonitis
- Mesenteric adenitis
- Constipation

## Patterns of pain

| Type of pathology | | | |
|---|---|---|---|
| **Inflammatory** | **Obstructive** | **Perforation** | **Visceral** |
| **Peritonitic pain**<br>→ fever, tachycardia<br>→ ↑WCC, ESR, CRp | **Colicky pain (waves of pain)**<br>→ vomiting, constipation<br>→ distension, tinkling sounds | **Sudden localised then peritonitic pain**<br>→ **shock** (↑HR, RR, ↓BP)<br>→ N&V | **Poorly localised pain (referred)**<br>→ specific Sx e.g. jaundice |

**Peritonitic pain** = worse on inspiration & movement → lie still, shallow breaths, **rigidity & guarding**

## Investigations & management

### INVESTIGATIONS:

- **Obs:** BP, HR, RR, sats, temp, ECG
- **Pregnancy test:** in all females of child-bearing age
- **Bloods:** FBC, U&Es, LFT, CRP, amylase, glucose, clotting, ABG (lactate)
- **Urinalysis:** protein, nitrates, leukocytes, blood, glucose, pH
- **Imaging:** USS, erect CXR, abdo CXR, CT
- **Specialist tests:** MRCP/ERCP, MRI, barium swallow, OGD, colonoscopy
  → **not always indicated**

### INITIAL MANAGEMENT: early senior input if concerned

- **ABCDE + targeted Mx of suspected cause**

**Things to consider:**

- Nutritional and feeding status – keep NBM if surgery likely soon or vomiting
- Hydration and fluid balance – IV or oral fluid maintenance, consider catheter
- Are antibiotics indicated? – signs consistent with infection
- Investigations to guide/aid management – blood cultures, ABG, G&S, CXR, ECG
- Is blood transfusion / major haemorrhage protocol required?
- Is theatre / surgical intervention needed? – NBM, consent, book theatre slot

### IMAGING INDICATIONS:

| X-ray | Abdo USS | CT |
|---|---|---|
| • Obstruction<br>• Toxic megacolon<br>• Foreign body | • Biliary pathologies<br>• Kidneys, ureter, bladder<br>• Gynae pathologies<br>• Appendix (in a female patient, no role in male) | • AAA/vascular<br>• Malignancy/mass<br>• Complications of obstructions<br>• Appendix if >50y |

### CONSIDERATIONS FOR SURGERY:

**Patient health:**

- Anaesthetic review
- Comorbidities/frailty
- **Ceiling of care:** ReSPECT form / DNACPR
- **Morbidity/mortality calculation** e.g. P-POSSUM score

**Patient wishes:**

- Current wishes
- Advance directives / ReSPECT forms
- Discussion with family

**NB:** the decision to operate is a complicated one and many factors should be considered.

See *Chapter 16: Critical illness* (and *Chapter 10: Anaesthetics* in the companion *Clinical Specialties* book) for further details.

**NB: Not all acute abdominal pathologies present with pain** – often absent in the elderly, children, diabetics & pregnant women

Amylase can be raised in some pathologies other than pancreatitis e.g. perforated duodenum

**Erect CXR:** may show air under diaphragm; **however**, a negative erect CXR does **not** exclude pneumoperitoneum

ReSPECT = Recommended Summary Plan for Emergency Care and Treatment, a patient-held form

# Acute pancreatitis

An **acute inflammatory reaction** due to **enzymes released** from damaged acini

↳ **Direct damage:** drugs/alcohol/infection → **panlobular damage**
↳ **Duct obstruction:** gallstones/tumour → **periductal damage**

## Causes: 'I GET SMASHED'

**I**diopathic (20%)

**G**allstones (40%)
**E**thanol (35%)
**T**rauma (15%)

**S**teroids
**M**umps (CMV, EBV)
**A**utoimmune (SLE, polyarteritis nodosa)
**S**corpion venom
**H**yperlipidaemia, **h**ypercalcaemia, **h**ypothermia
**E**RCP
**D**rugs (thiazides, sulphonamides, ACEis, NSAIDs)

**Symptoms:**

- Severe **epigastric / upper abdo pain**
  → radiates to back
  → better sitting forward
- Nausea & vomiting

**Signs:**

- Fever, tachycardia
- Jaundice (in 30%)
- Oedema ± hypovolaemia
- **Cullen & Grey Turner signs**

Very late signs

## Investigations

- **History & examination** – drugs, alcohol, infection
- **Routine bloods:** FBC, CRP/ESR, U&Es, LFTs, clotting, calcium, lipids + pregnancy test
- **Serum amylase** or **serum lipase** – depending on what is available at your local Trust
- **ABG:** monitor acid–base status
- **Erect CXR:** exclude perforation
- **Normal CXR:** check for ARDS (a possible complication)
- **Other imaging:**
  - USS – if suspect gallstones / no obvious cause
  - MRCP/CT – detailed view of severity / extent of damage
  - Endoscopic USS – if other imaging negative

**lipase** = more **sensitive & specific** → amylase also ↑ in GI perfs & mesenteric infarct

**amylase = very sensitive in first 24h** then levels start to drop

**AXR** only done if looking for additional pathology, but would show:

- loss of psoas shadow (due to retroperitoneal haemorrhage)
- sentinel loop of jejunum (air-filled dilation due to ileus)

## Determining severity of acute pancreatitis

**1. Modified Glasgow Score (MGS) / 'PANCREAS' score**

| | |
|---|---|
| **P** | ***$PaO_2$*** <8kPa |
| **A** | **Age** >55y |
| **N** | Neutrophils: **WCC** $>15\times10^9$ |
| **C** | **Calcium** <2mmol/L |
| **R** | Renal: **urea** >16mmol/L |
| **E** | Enzymes: **LDH**>600 or **AST**>200 |
| **A** | **Albumin** <32g/L |
| **S** | Sugar: **glucose** >10mmol/L |

**Score ≥3 = severe**
Discuss with ICU/HDU →

**Ranson's criteria:**
Scores similar parameters

**2. APACHE II score**
Assesses disease severity in patients admitted to ICU
**Considers:** clinical parameters, age & comorbidities

## Management of acute pancreatitis[1]

1. **ABCDE:** IV fluids, $O_2$, catheter, analgesia
2. **Identify & treat cause:** e.g. cholecystectomy for gallstones
3. **Close monitoring:** FBC, U&Es, Ca, glucose, ABG (CRP = good indicator of progression)
4. **Consider feeding status:** only keep NBM if very unwell (consider NG tube)

**Prognosis**
**85% settle in 5–7d**
15% need ICU admission
Early organ failure = poor prognostic factor

## Complications of acute pancreatitis

| Early | Late |
|---|---|
| Pain<br>Hypovolaemic shock → renal failure<br>Hyperglycaemia/hypocalcaemia<br>DIC & sepsis<br>Acute respiratory distress syndrome | Pseudocyst<br>Abscess<br>Splenic/duodenal/SMA infarct<br>Chronic pancreatitis → cancer |

**PANCREATIC PSEUDOCYST** = **collection of necrotic tissue & fluid forming 4–6w after acute pancreatitis**

**Symptoms:**

- Epigastric pain = deep & persistent
- N&V, anorexia, weight loss

**Investigations:**

- CT abdo = gold standard
- ± ERCP (to plan therapy)

**Management:**

1. **Most = self-resolving** → supportive care & monitoring
2. **Drainage:** percutaneous, endoscopic (ERCP), laparoscopic or open

**Indications for drainage:**

- Symptomatic relief
- Complications (bleeding/infection)
- Concern about malignancy

---

[1] NICE (2018, updated 2020) *Pancreatitis* [NG104]

# Meckel’s diverticulum

**Rule of 2s**

- 2% of population
- 2 years old = peak presentation
- 2:1 ratio of M:F
- 2 inches long
- 2 feet proximal to ileocaecal valve
- 2 types of ectopic tissue (gastric/pancreatic)

Remnant of embryological **vitello-intestinal duct**

## Clinical presentation / complications

→ *most are* ***asymptomatic***

- **Intussusception:** common around 2y (painless melaena + obstruction)
- **Caecal volvulus:** obstruction & distension
- **Peptic ulceration:** epigastric pain with eating
- **General inflammation:** presents like **acute appendicitis**

## Investigations

→ *usually an incidental finding at diagnostic laparoscopy*

→ **Technetium scan:** if gastric mucosa (parietal cells take up radioisotope)

→ **CT:** shows defect in abdo wall

## Management

**If asymptomatic:** no intervention → otherwise surgical removal (depending on presenting complaint)

# Acute appendicitis

= most common surgical emergency

## Pathology

→ **Luminal obstruction due to:** faecolith, FB or enlarged LNs
→ **Bacteria proliferate** in closed loop of bowel
→ Swelling → Ischaemia → Necrosis → Perforation

## Clinical presentation

- Vague periumbilical pain moves to **localise in RIF** (**McBurney's point**)
- Nausea & anorexia ± vomiting & diarrhoea

## On examination

- Rebound & percussion tenderness
- Guarding
- **+ve Rovsing's sign** – pain in RIF when LIF is pressed
- **+ve psoas sign** – pain on right hip extension (retrocaecal appendix)
- **+ve obturator sign** – pain on internal hip rotation (referred knee pain)

± fever, tachycardia, tachypnoea

*** APPENDICITIS = CLINICAL DIAGNOSIS ***

## Investigations

**Hx & examination:** abdo (± PR exam ± pelvic exam in females if indication like PV bleeding)
**Bedside:** basic obs, urine dip, blood glucose, **pregnancy test** (+ ECG if cardiac history or >40y and needing surgery)
**Bloods:** ↑ WCC, CRP (remember amylase, U&Es, LFTs to r/o other causes of acute abdo)
**Imaging:**

- USS – not always able to visualise appendix but can r/o other pathologies in females / paediatric patients
- CT – if still unsure of Dx or anyone aged >50y to r/o malignancy

## Management[2]

**Acute:** ABCDE (IV ABX as per local guidelines) + urgent surgical review

**Definitive: appendicectomy** (within 24h)

- Pre-op ABX (as per local guidelines)
- Consider if need to be NBM
- Laparoscopic approach unless contraindicated
- Appendix sent for histology once removed

Consider non-operative management (IV ABX) in uncomplicated cases with absence of appendicolith

## Complications of untreated appendicitis

- **Perforated appendix**
- **Peritonitis/sepsis**
- **Appendix abscess:** can spread up paracolic gutter
- **Appendix mass** – inflamed appendix becomes covered in fibrotic omentum

**Differentials:**

- Diverticulitis/Meckel's
- Mesenteric lymphadenitis
- Cystitis/pyelonephritis/UTI
- Cholecystitis
- IBD
- **Ectopic pregnancy**
- Ovarian cyst rupture
- Testicular/ovarian torsion

**McBurney's point:** 1/3 of the way between anterior, superior iliac spine & umbilicus

**Variable presentation:**

- **Elderly:** shocked/confused
- **Children:** vague pain + anorexia
- **Pregnancy:** ↑ risk of perforation

must r/o ectopic in all women of childbearing age

**DDx of mass in RIF: Usually needs CT for diagnosis**

- **Crohn's:** fibrosis & thickening
- **Tumour:** caecal/colonic
- **Inflammatory:** appendix mass, abscess, TB granuloma
- **Ovarian cyst**
- **Psoas abscess**
- **Pelvic kidney** – transplanted kidney placed in RIF

**Management of appendix mass / abscess**

1. **Conservative:** IV fluids + ABX
2. IR-guided drain (if suitable)
3. Delayed appendicectomy

---

[2] Di Saverio *et al*. (2020) Diagnosis and treatment of acute appendicitis. *World J Emerg Surg*, 15:27

# Diverticular disease

Affects 50% of those over 50y

→ **Diverticulum:** outpouching of bowel wall (at areas of high pressure)
→ **Diverticulosis:** asymptomatic presence of diverticula
→ **Diverticular disease:** diverticulosis + **symptoms**
→ **Diverticulitis:** inflammation of diverticula (due to stagnated contents)

## Pathology

Weakened bowel → stool passage ↑s pressure → outpouching

Often at sigmoid colon

## Risk factors[3]

- **Diet:** low fibre, high fat → **constipation**
- **Age** >50y & **male gender**
- **Obesity**
- **CTD** e.g. Marfan, Ehlers–Danlos syndrome (weak bowel wall)

## Diverticular disease vs. diverticulitis

| | Diverticular disease | Diverticulitis |
|---|---|---|
| **Features** | • **Left-sided colicky pain** – relief with defecation<br>• **Altered bowel habit**<br>• **Sudden painless bleed*** – bright red<br>→ **mimics colorectal cancer** | • **Severe LIF pain** – acute, worse with movement<br>• **Localised guarding & tenderness** – in LIF<br>• **Systemic upset:** fever, ↑ HR, N&V<br>→ **Hinchey Class:** 1–4 (small abscess to free faeces) |
| **Investigations** | **Bedside:** obs, abdo, DRE, urine dip = **normal**<br>**Bloods:** FBC, U&Es, LFTs = **normal**<br>**Imaging:** flexible sigmoid-/colonoscopy = **diagnostic** (& r/o cancer)<br>**Risks of endoscopy:**<br>• perforation<br>• haemorrhage<br>• infection | **Bedside:** obs, abdo, DRE, urine dip = ↑ HR & temp<br>**Bloods:** FBC, U&Es, LFTs + blood cultures + ABG = ↑ WCC, CRP & ESR<br>**Imaging:**<br>**1.** CXR = pneumoperitoneum if perforated<br>**2.** AXR = check for obstruction<br>**3.** CT abdo/pelvis = **diagnostic**<br>→ **Avoid endoscopy in acute phase** as risk of perforation |
| **Management[3]** | **Conservative:**<br>• Fluids & fibre<br>• Smoking cessation & weight loss<br>**Medical:**<br>• Analgesia (paracetamol)<br>• Bulk-forming laxatives | **Admission: ABCDE**<br>• Analgesia (paracetamol)<br>• Broad-spectrum IV ABX (if systemically unwell)<br>• IV fluids only if cannot tolerate oral intake<br>**Surgery:** if complications e.g. perforation<br>→ Hartmann's procedure |

**Complications of diverticular disease:**
- Perforation → peritonitic, very unwell (abdo sepsis)
- Obstruction → constipation, distension, vomiting
- Fistula → pneumaturia, bladder/uterus infection
- Pericolic abscess → pus within bowel wall (fever)

*bleed if erodes vessel

**DDx of diverticular disease:**
- **Colorectal cancer**
- **IBD** (or IBS)
- Ischaemic colitis
- Gynae cause: ruptured cyst, ectopic, PID
- Renal stones

**Diverticular bleeds:** if erodes vessel

**Sx:** **large, painless bleed** → hypovolaemic shock
**Signs:** ↑ HR, ↑ RR, ↓ BP, ↑ WCC & CRP, ↓ Hb
**Ix:** CXR & AXR (r/o perforation & obstruction)
**Mx:**
1. ABCDE & urgent colonoscopy / CT angio to find source
2. May need blood transfusion

---

[3] NICE (2019) *Diverticular disease* [NG147]

# Gastrointestinal perforation

Loss of continuity of the gastrointestinal tract wall, allowing contents to leak into the abdominal cavity

## Causes

- **Ischaemia** (bowel obstruction causing necrosis)
- **Infection** (appendicitis, diverticulitis)
- **Erosion** (ulcers, malignancy)
- **Direct damage** (trauma, instrumentation/iatrogenic)

## Symptoms

- **Abdominal pain** – peritonitis (guarding, rigidity)
- Nausea & vomiting
- Reduced bowel movements / constipation
- ± Fever ± septic shock

## Investigations

- AXR – can show bowel obstruction
- Erect CXR – may show pneumoperitoneum
- Barium swallow – assess for UGI perforation
- CTAP – most sensitive and specific

Absence of pneumoperitoneum **does not** rule out abdominal organ perforation

## Management

**Urgent surgical referral**

1. **Supportive** – bowel rest, IV fluids, NG tube, analgesia, catheter
2. **Medical** – IV ABX
3. **Non-operative** – collections drained by interventional radiology
4. **Surgical intervention** – procedure depends on type of perforation

# Intestinal obstruction

→ 10% of all surgical admissions

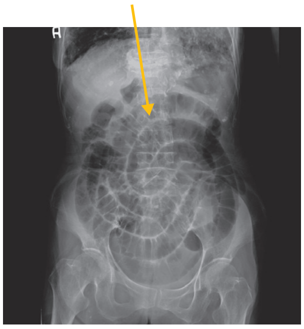
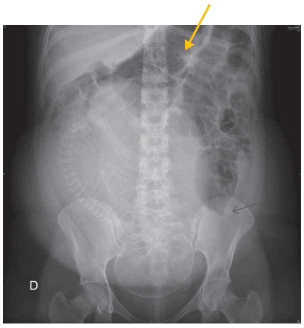
*Paralytic ileus = not true obstruction, but can present with similar symptoms

## Types

1. **Paralytic ileus***: functional problem of **absent GI motility** (no colicky pain)
   - systemic infections
   - neurological disorders
   - metabolic disturbance
   - **recent surgery** (inflammation & opioids)

2. **Mechanical obstruction:** blockage of passage (intrinsic, extrinsic, intramural)
   distal collapse & proximal dilation of bowel
   congestion & oedema → ischaemia → necrosis

## Overview of mechanical obstruction

| | Small bowel | Large bowel |
|---|---|---|
| **Causes** | • **Adhesions** (scar tissue)<br>• **Hernias**<br>• Crohn's (strictures)<br>• Intussusception (<3y)<br>• Gallstones/CF<br>• Malignancy (rare)<br>**Risk factors:**<br>• Previous surgery<br>• IBD<br>• Biliary disease | • **Colorectal cancer**<br>• **Diverticulitis**<br>• Crohn's/UC<br>• Faecal impaction (elderly)<br>• Volvulus<br>• (Adhesions/hernias) |
| **Symptoms** | • **Colicky pain**<br>• **Vomiting** (prominent, **green**)<br>• Constipation | • **Abdo pain** (± colicky)<br>• **Constipation** (prominent & early)<br>• Vomiting |
| **Examination** | Distension (mild) Dehydration (↑ urea) Tinkling bowel sounds | |
| | **Signs of the cause: very important to note**<br>• Hernias<br>• Abdominal scars<br>• History of IBD | **Signs of the cause: very important to note**<br>• **Red flags for colorectal cancer (weight loss, PR bleed, IDA)** |
| **Investigations** | **1. History:**<br>• Risk factors (red flags for cancer, previous surgery, IBD)<br>• Symptoms (last bowel movement / flatus, N&V)<br>**2. Bloods:**<br>• FBC, CRP (raised inflammatory markers, presence of anaemia)<br>• U&Es – deranged (electrolyte disturbance)<br>• Amylase, LFTs, ABG<br>**3. Imaging:**<br>• Abdo XR<br>• Erect CXR (may show free air if perforation)<br>• CT abdo + pelvis (shows site & cause) | |
| **Radiology** | → **Central** dilation<br>→ 3–5cm<br>→ **Valvulae conniventes** (cross whole width)<br><br>Fig. 11.2 | → **Peripheral** dilation<br>→ >5cm<br>→ **Haustra** (do not cross whole width)<br><br>Fig. 11.3 |

| | Small bowel | Large bowel |
|---|---|---|
| **Management**[4] | **'Drip & suck'**<br>1. **Fluid resuscitation** – IV drip + catheter<br>2. **Decompression** – NG tube<br>+ IV ABX if suspected sepsis<br>**Surgery if:**<br>→ No improvement in 48h<br>→ Irreducible hernia<br>→ Suspected perforation or ischaemia<br>→ No previous surgical Hx to suggest adhesions<br>If adhesions suspected, may try water-soluble contrast enema before surgical intervention | **'Drip & suck'**<br>1. **Fluid resuscitation** – IV drip + catheter<br>2. **Decompression** – NG tube<br>+ IV ABX if suspected sepsis<br>**Surgery usually needed:**<br>**Stent:** may be possible to relieve obstruction, depending on site & cause<br>**Colostomy:** exact procedure depends on location of obstruction<br>**If sigmoid volvulus: rectal insufflation** (flatus tube with gas enema pumped in) |

## Complications of bowel obstruction

1. **Fluid & electrolyte shifts** → dehydration, metabolic acidosis & hypovolaemic shock
2. **Strangulation** → pain becomes severe & constant ± fever, tachycardia etc.
3. **Perforation** → peritonitic pain ± fever, tachycardia, raised inflammatory markers

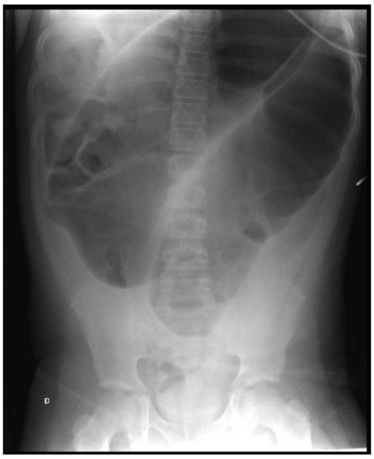


**Fig. 11.4** Sigmoid volvulus ('coffee bean' shape).

**Assess competence of ileocaecal valve**

No air in small bowel = competent valve = closed loop so ↑ **risk of perforation**

### Faecal impaction

**Risk factors:** low fibre diet, dehydration, immobility (common in elderly)

**Management:** fluid resuscitation, suppository/enema, laxatives / stool softeners → **manual evacuation if needed**

### Causes of paediatric bowel obstruction:

- Intussusception
- Faecal impaction
- Hernia
- Malrotation/atresia
- Hirschsprung's disease
- Pyloric stenosis
- Adhesions
- Imperforate anus

---

[4] Association of Surgeons of Great Britain and Ireland (2014) *Commissioning guide emergency general surgery*

# Hernias

= protrusion of an organ through a normal/abnormal opening in the wall

Usually a painless lump

Obstructed hernia = if bowel contents trapped

- Abdo pain & distension
- Constipation
- N&V

**Deep ring:** 1cm superior to midpoint of inguinal ligament

**Superficial ring:** 1cm superior and lateral to pubic tubercle

**Deep ring occlusion test: differentiate direct & indirect hernias**

- Press on deep ring to reduce hernia
- Get patient to cough
- If hernia reappears = **LIKELY DIRECT**

**USS for definitive Dx**

**Elective surgical options:**

**Lichtenstein open repair:**
→ mesh repair under local or general anaesthetic

**Laparoscopic repair:**
→ if bilateral/recurrent hernia and experienced surgeon available

## Classification

- **Reducible:** can be manipulated back through defect
- **Irreducible:** cannot reduce without surgery
- **Incarcerated:** irreducible hernia with contents trapped
- **Strangulated:** ischaemia of contents → necrosis → sepsis = **emergency**
  - Sudden intense pain
  - Red/purple/blue swelling = **irreducible**

## Inguinal hernia

↳ *superomedial to pubic tubercle → most common type*

### ANATOMY OF THE INGUINAL CANAL:

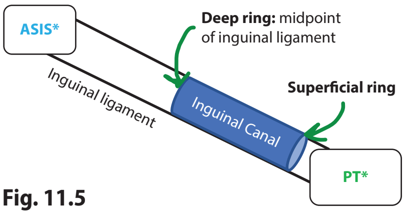


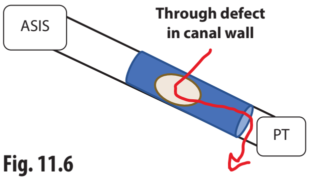
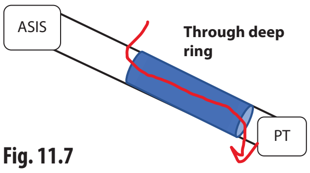
**Fig. 11.5**

***ASIS:** Anterior, superior iliac spine
***PT:** Pubic tubercle

### CONTENTS OF INGUINAL CANAL:

- **3 arteries:** testicular/ovarian, vas deferens, cremasteric
- **3 nerves:** ilioinguinal, genitofemoral, sympathetic
- **3 others:** vas deferens / round ligament, pampiniform plexus, lymphatics

### TYPES OF INGUINAL HERNIA:

| | Direct (1/3) | Indirect (2/3) |
|---|---|---|
| **What?** |  <br>**Fig. 11.6** |  <br>**Fig. 11.7** |
| **Why?** | **Acquired** (weakened wall + ↑ intra-abdominal pressure)<br>**RF** = chronic coughing, constipation, obesity, heavy lifting, protein deficiency (elderly) | **Congenital**<br>(incomplete obliteration of processus vaginalis) |
| **Who?** | Older men | Younger patients (especially boys) |
| **Features** | → **Easily reducible**<br>→ Rarely extends to scrotum<br>→ Rarely strangulates | → **Harder to reduce**<br>→ May extend to scrotum<br>→ More likely to strangulate |

### MANAGEMENT OF INGUINAL HERNIA:

1. **Reducible & asymptomatic:** none
2. **Symptomatic:** consider elective surgery
3. **Acutely irreducible / obstructed / strangulated:** emergency surgery
   - **CT abdo** to demonstrate contents of hernia & viability of bowel
   - NG tube, IV fluids & catheter **if obstructed**
   - **Surgery:** groin incision ± laparotomy ± bowel resection

## Femoral hernia

*→ inferolateral to pubic tubercle (uncommon)*

USS/CT for definitive Dx

### ANATOMY OF THE FEMORAL CANAL:

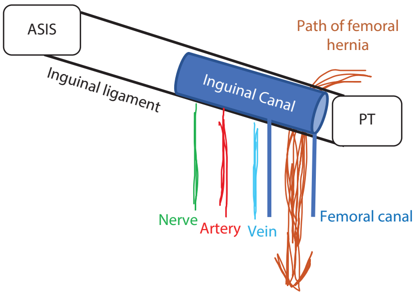


**Fig. 11.8**

**Femoral canal:**

- Medial to vessels
- Allows expansion of vein

**Femoral hernia:**

- Abdominal viscera / omentum passes through femoral canal

**MANAGEMENT:** **operate on all within 2w of diagnosis**

- Reduce hernia & narrow femoral ring with sutures or mesh plug

**Risk factors for femoral hernia:**

- **Female**
- Pregnancy/multiparous
- ↑ Intra-abdominal pressure

Femoral hernias = **high risk of strangulation**
30% present as an emergency

**Operate on all within 2w**

**DDx of groin lumps:**

- Inguinal hernia
- Femoral hernia
- Lymph nodes
- Lipoma
- Ovarian cyst
- Femoral artery / iliac artery aneurysm
- Saphena varix
- Appendicular mass
- Appendicular abscess
- Tumour

*Must examine regional lymph nodes for any groin lump*

## Richter's hernia

= Hernia of the bowel that **only involves the bowel wall** and **not the lumen**

- Most common at **femoral ring**
- May strangulate/perforate **without any warning signs of obstruction**

## Umbilical & paraumbilical hernias

| | Umbilical | Paraumbilical |
|---|---|---|
| **Aetiology** | Herniation of bowel through **umbilical ring** due to defect in **transversalis fascia** | Herniation of bowel through **weakness in linea alba** due to ↑ **IAP** (just above/below umbilicus) |
| **Who?** | Congenital (usually children) | Women aged 35–50y |
| **Risk factors** | Black, male, premature | Obesity, multips, heavy lifting / chronic cough |
| **Features** | → Easily reducible<br>→ **Usually asymptomatic**<br>→ Rarely strangulate | → May become irreducible<br>→ **Intermittent colicky pain** (bowel obstruction)<br>→ **Higher risk of strangulation if small defect** |
| **Management** | 90% close spontaneously by 2y | Early surgery before obstruction |

## Epigastric hernia

*→ herniation of fat through linea alba above umbilicus*

**Symptoms:**

- **Painful** abdominal mass (worse with eating)
- ± N&V, bloating

**Management:**

- Surgery if symptomatic (as this suggests strangulation)

**DDx:**

- Divarication of recti

**Risk factors for incisional hernia**

| Patient RFs | Surgical RFs |
|---|---|
| • ↑ age<br>• Malnutrition<br>• Obesity<br>• Diabetes<br>• Steroids<br>• Smoking | • Vertical incisions<br>• Presence of drains<br>• Too loose or too tight knots<br>• Emergency surgery<br>• Wound infection |

## Divarication of recti

→ *separation of rectus muscles due to linea alba laxity*

**Risk factors:** truncal obesity, pregnancy, repeated midline operations
**Symptoms:** **painless** ridge down midline
**Ix:** USS to r/o hernia
**Management:** not usually needed

## Incisional hernia

→ *herniation through a previous incision/scar in abdominal wall*

**Who:** common risk after any abdominal surgery
**Symptoms:** bulge at scar site, local pain/erythema → can obstruct
**Investigation if repairing:** CT scan
**Management:** none / mesh repair → **but high recurrence rate**

**Prevalence:**
→ 5% at 1y
→ 25% at 2y

**Indications for repair:**
Large, patient Sx, small bowel obstruction

↳ **RFs for recurrence:**
- Obesity
- Chronic cough

## General approach to managing a hernia

**1. HISTORY:**
→ **PCx:** site, onset, duration, any pain
→ **PMHx:** PSHx, DHx (especially drugs which may affect surgery)
→ **SHx:** smoking/alcohol, BMI, independence / help at home

**2. EXAMINE:** remember to check for other hernias

If unsure of Dx can use USS or CT scan

→ General appearance
→ Observe standing & lying
→ Palpate hernia (get patient to cough & lift chin off bed) → reducible/irreducible?
→ Deep ring occlusion test → direct / indirect?

**3. SURGERY:** decide if appropriate
→ depends on type/size/symptoms of hernia & fitness of patient

# GASTROINTESTINAL SURGERY

# 12

**Perianal disease** .......... 166
**Colorectal cancer** .......... 168
**Anal cancer** .......... 170

## ABBREVIATIONS

**CEA** – Carcinoembryonic antigen
**CRC** – Colorectal cancer
**CT CAP** – Computed tomography chest, abdomen & pelvis
**DM** – Diabetes mellitus
**DRE** – Digital rectal examination
**EUA** – Examination under anaesthetic
**FAP** – Familial adenomatous polyposis
**GA** – General anaesthesia
**GTN** – Glyceryl trinitrate
**HNPCC** – Hereditary non-polyposis colorectal cancer
**HSV** – Herpes simplex virus
**IAP** – Intra-abdominal pressure
**IBD** – Inflammatory bowel disease
**IDA** – Iron-deficiency anaemia
**LN** – Lymph node
**PR** – Per rectum
**PV** – Per vagina
**SB** – Small bowel
**UC** – Ulcerative colitis
**UTI** – Urinary tract infection

# Perianal disease

→ **Infections commonly occur in anal crypts/glands**
→ symptoms of fever, pain, swelling, discharge

**DDx perianal lump:**

- Abscess
- Pilonidal sinus
- Fistula-in-ano
- Anal fissure
- Sebaceous cyst
- Crohn's disease
- Thrombosed external haemorrhoid

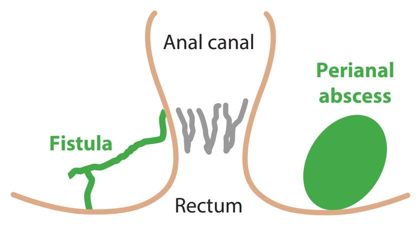


**Fig. 12.1**

**Conditions predisposing to fistula-in-ano:**

- Crohn's
- TB
- Diverticular disease
- Malignancy

**Conditions predisposing to anal fissure:**

- Crohn's
- Anal cancer
- HIV, HSV, syphilis
- **Hard faeces**

## Fistulae

**DEFINITION:** abnormal connection between 2 mucosal surfaces

**TYPES:** SB = small bowel

**Intrinsic:** entero-enteric (SB–SB) / entero-colonic (SB–colon) / entero-vesical (SB–bladder)

**Extrinsic:** entero-cutaneous (SB–skin) / entero-vaginal (SB–vagina) / recto-vaginal etc.

**SYMPTOMS:**

**To skin:** discharging sinus or abscess
**Bowel–bowel:** nothing / diarrhoea / weight loss / pain
**To bladder:** pneumaturia, debris in urine, recurrent UTIs
**To vagina:** PV discharge

**FISTULA-IN-ANO** = tract between rectum / anal canal & skin

**Symptoms:** discharging tract + recurrent perianal infection

**Investigations:**

- Acute perianal pain: examination under anaesthetic (EUA)
- Chronic/complex presentation: MRI

**Management:**

1. **Very superficial** – watch and wait
2. **Superficial/intersphincteric:** lay open (fistulotomy) → ABX → leave to heal by 2° intention
3. **Higher level (involves external sphincter):** ABX + seton suture drainage

*Seton suture:* piece of thread remains in fistula tract to keep it open & allow drainage → gradually tightened to enable healing & closure

## Perianal abscess

**SYMPTOMS:** red, sore, swollen lesion (± discharge)

Abscess usually contains gut organisms: E. coli, bacteroides, enterococcus

**ASSOCIATIONS:** Crohn's, DM, malignancy, anal sex

**MANAGEMENT:** incision & drainage (under GA) → ABX → apply dressings
→ **Aim to treat before ruptures & discharges to form fistula**

## Anal fissure

**DEFINITION:** tear in squamous lining of anal canal **below dentate line**

**AETIOLOGY:**

- **90%** = posterior to anus
- **10%** = anterior to anus

**SYMPTOMS:**

- **Very painful** → esp. defecating & for hours after **(cannot tolerate PR exam)**
- Bleeding on defecation (bright red)
- Constipation (avoid defecating due to pain)
- Pruritus ani
- Mucosal skin tag / sentinel pile

**MANAGEMENT[1]:**

1. **Conservative:** fluids & fibre, bulking laxatives, lubricants, **topical analgesia**
2. **Fissures unresponsive to conservative measures:** topical GTN cream for 6–8w
3. **Unresponsive to medical management:** EUA ± further management
   → Botox or sphincterotomy = last resort

---

[1] NICE CKS Clinical scenario (2021) *Anal fissure*

## Haemorrhoids

**DEFINITION:** distension & prolapse of **normal anal cushions** (masses of spongy, venous tissue)

**CAUSES/AETIOLOGY:**

- Idiopathic – weakened muscular wall with age
- Chronic constipation/straining (low fibre diet)
- Raised IAP (pregnancy, portal HTN, pelvic tumour)

**CLINICAL PRESENTATION:**

- **Painless, bright red PR bleed** (coats stool & dripping into pan)
- Itching
- Mucous discharge

→ **If thrombosed:** painful & tender, purple mass

**MANAGEMENT[2]:**

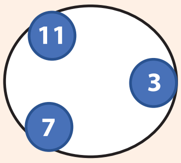
Asymptomatic haemorrhoids require no further investigation

1. **Conservative:** fluid & fibre, laxatives, topical analgesia, toileting advice (correct position, avoid straining)
2. **Non-operative:** rubber band ligation / injectable sclerosants
3. **Surgical:** haemorrhoid artery ligation / haemorrhoidectomy

→ **Management of acutely painful thrombosed haemorrhoid:** analgesia + ice pack + Instillagel + laxative

↳ No role for surgery

**Cushions found at 3, 7 & 11 o'clock**



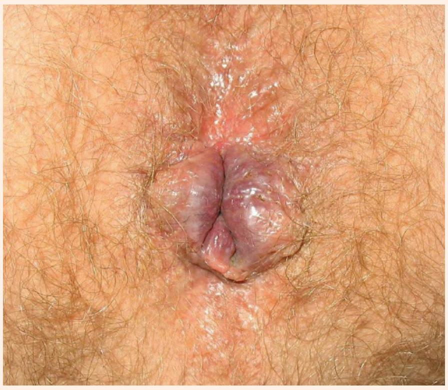


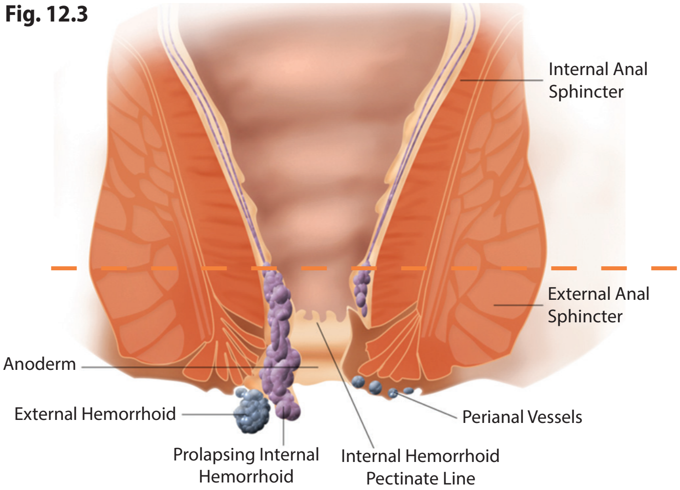
**Fig. 12.2** External haemorrhoid.

**Investigations:**

- **DRE** + abdo exam
- Proctoscopy (shows pink mucosal swellings)
- Flexible sigmoid-/colonoscopy (r/o sinister bleed)

**ANATOMY:**

**Fig. 12.3**



**Internal haemorrhoids** = above dentate line

→ **Epithelium:** columnar
→ **Venous drainage:** portal system
→ **Somatic innervation:** none = painless

**External haemorrhoids** = below dentate line

→ **Epithelium:** squamous
→ **Venous drainage:** internal iliac
→ **Somatic innervation:** present = painful if thrombosed or become irritated

## DDx of PR bleed

| | With pain | Without pain* |
|---|---|---|
| **Blood mixed with stool** | Colitis | Colorectal cancer / colitis |
| **Blood streaked on stool** | Anal tumour | Rectal tumour |
| **Blood separated from stool** | Colitis | Haemorrhoids<br>Diverticular disease<br>Angiodysplasia<br>Tumour<br>Colitis (with mucus) |
| **Blood streaked on paper** | Anal fissure | Haemorrhoids |

*Sometimes these can present with pain as well!

**Important questions to ask:**

- **How long?**
- **How much?**
- **Mixed with stool?** (above colon = usually mixed in)
- **Pain?**
- **Altered bowel habits?** (compare to normal for the patient)
- **FHX / personal Hx**

→ **check red flag symptoms** (see *Colorectal cancer* on next page)

---

[2] European Society of ColoProctology (2020) *Guideline for haemorrhoidal disease*

# Colorectal cancer

↳ most = adenocarcinomas

→ **3rd most common cancer**
→ 2nd most common cause of cancer deaths
→ **Peak age:** >60y
→ M>F
→ **1 in 30 risk**

### Familial adenomatous polyposis (FAP):

Auto dominant mutation in *APC* gene

→ **Hundreds of polyps**
→ Presents at **35–45y** (consider if <50y with colorectal cancer)
→ Colonoscopy every **2y** from **25 to 75y**

### HNPCC / Lynch syndrome:

Auto dominant mutation in DNA repair genes

→ Cancers of colon, endometrium, ovary, stomach, bladder, brain, skin
→ Presents **>40y** (consider if <50y with colorectal cancer)
→ Colonoscopy every **2y** from **25 to 75y**

NB: non-polyposis condition (no polyps present)

Amsterdam criteria to Dx

### Bowel cancer screening programme[3]:

**qFIT (quantifiable faecal immunochemical test)**
→ if >120mcg/g refer for colonoscopy to remove any polyps

Every 2y from the age of 50y

### May present as an acute emergency:

- Bowel obstruction
- Bowel perforation
- Extreme pain

### Red flags for 2ww referral[4]:

1. ≥40y with unexplained weight loss & abdo pain
2. ≥50y with unexplained rectal bleeding
3. ≥60y with altered bowel habit **or** IDA
4. Occult blood present when faeces tested

Also consider if:

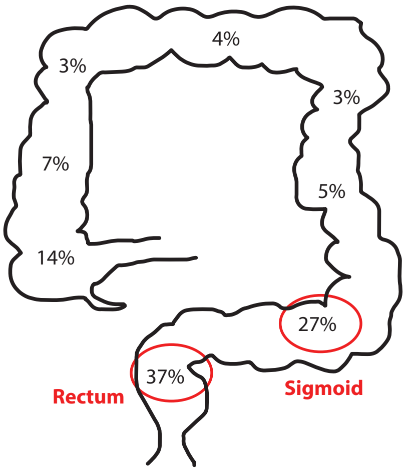
- Rectal or abdominal mass
- ≤50y with rectal bleeding and any of:
  - Abdominal pain
  - Altered bowel habit
  - Weight loss
  - IDA

## Risk factors

- **Polyps** (UC, HNPCC, FAP) → identify & remove
- **Age** (90% are aged >50 y)
- **Diet** – low fibre, high fat, processed meat
- **Smoking/alcohol/obesity**
- **IBD** (UC especially)
- **Genetics** (FAP & HNPCC / Lynch syndrome)
- **FHx**

**Polypectomy + follow-up endoscopy** (complications = bleeding & perforation)

## Clinical presentation



Liver = common site of colorectal cancer metastases

**Fig. 12.4** Sites of colorectal cancer.

| Right side | Left side | Rectal |
|---|---|---|
| = Late presentation<br>• Weight loss / fatigue<br>• IDA<br>± Abdo pain/mass | • Weight loss / fatigue<br>• Change in bowel habit<br>• Obstruction<br>• Blood-streaked stools<br>± Abdo pain/mass | • Weight loss / fatigue<br>• Tenesmus<br>• Bright PR bleed<br>• **DRE:** palpable mass |

→ 20% present with signs of **disseminated disease** e.g. liver mets = jaundice

## Investigations

1. **DRE**
2. **Bloods** – FBC (for IDA), LFTs, U&Es
3. **Tumour marker:** CEA (not specific – use for monitoring)
4. **Colonoscopy & biopsy = gold standard**
5. **MRI** – if rectal cancer
6. **CT (chest, abdo, pelvis)** – for TNM staging

## Classification & staging

**Duke's criteria** or **TNM staging**

| Stage | Duke's stage | Features | 5y survival[5] |
|---|---|---|---|
| 1 | A | Mucosa only | 90–95% |
| 2 | B1 | Into muscularis | 80–85% |
| | B2 | Through wall | |
| 3 | C1 | T2 + nodes | 60–65% |
| | C2 | T3 + nodes | |
| 4 | D | Distant mets | <11% |

---

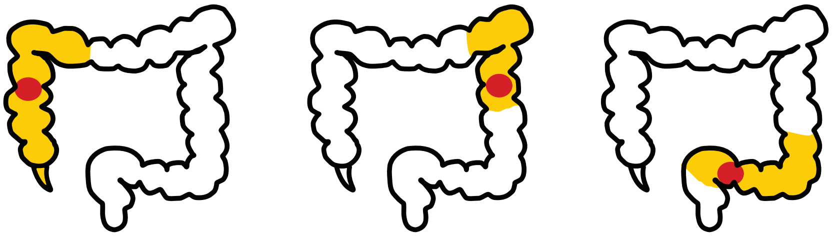
[3] Bowel cancer screening programme (www.gov.uk/guidance/bowel-cancer-screening-programme-overview)
[4] NICE (2015, updated 2021) *Suspected cancer: recognition and referral* [NG12]
[5] Cancer survival by stage at diagnosis for England, 2019. ONS.

## Management of colorectal cancer[6]

→ **colectomy + 'en bloc' lymph node removal** ± chemotherapy

**Different levels of resection:** ● = location of tumour

**Left hemicolectomy** **Right hemicolectomy** **Sigmoid colectomy**



**Anterior resection** **Abdominoperineal resection** **Panproctocolectomy**

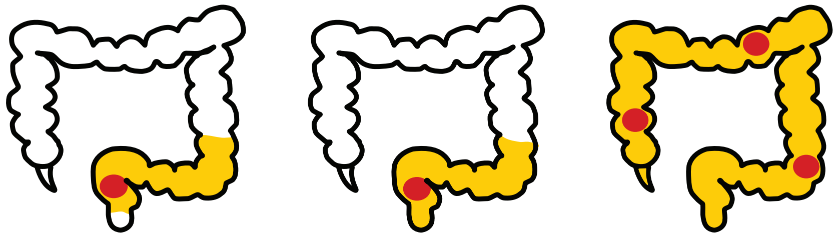


Fig. 12.5

### MANAGEMENT OF OBSTRUCTING COLON CANCER: emergency

1. ABCDE
2. Analgesia + NG tube decompression
3. CT scan if no small bowel distension = competent ileocaecal valve = BAD
4. Endoscopic stenting **or** colectomy (often with stoma) → colectomy type depends on obstruction location

## Management of rectal cancer[6]

1. **Total mesorectal excision** (removal of rectum)
    - **Low tumours <5cm above anus:** abdominoperineal excision (resect + stoma)
    - **Higher tumours >5cm from anus:** anterior resection (resect + anastomosis)
2. **± Chemotherapy ± radiotherapy**

## Colorectal cancer follow-up

**Follow-up for 5y post-surgery**

1. **CEA** – check every 6m
2. **CT CAP** – annually
3. **Colonoscopy** – at 1 & 5y

**20–30% recurrence**

**MDT input**
including stoma nurse / specialist nurse

**Lymph drainage:**

→ Ascending & transverse drain to **superior mesenteric LNs**
→ Descending & sigmoid drain to **inferior mesenteric LNs**
→ Rectum (above pectinate line) to **internal iliac LNs**

**Hartmann's**
= sigmoid colectomy with colostomy (stoma formation)

**Panproctocolectomy**
= if synchronous cancer or inflammatory bowel disease

**Stoma formation:**

Elective or emergency

**Decision depends on:**

- location of resection
- indication for operation
- patient comorbidities
- patient lifestyle/preference

---

[6] NICE (2020, updated 2021) *Colorectal cancer* [NG151]

# Anal cancer

**Methods of spread:**

1. **Local invasion** of rectum, scrotum, vagina
2. **LN drainage →** to **inguinal lymph nodes**

## Aetiology

→ **RARE** (associated with HIV & HPV infection)
→ 80% = **SQUAMOUS CELL**

## Clinical presentation

- Anal pain / pruritus
- Anal bleeding/discharge
- Mass/ulcer
- Incontinence
- Fistula-in-ano

## Investigations

- **EUA**
- HIV testing
- CT chest, abdo, pelvis – for mets
- MRI – assess local spread
- PET – assess LN involvement

## Staging

**NB: Different T staging to colorectal cancer** (based on size)

| | |
|---|---|
| **T1** | <2cm |
| **T2** | <4cm |
| **T3** | >4cm |
| **T4a** | Invades vagina |
| **T4b** | Invades other structures |

## Management

1. **Small tumours at margin** – local excision
2. **All others:** chemoradiotherapy (60–70% success)

# THE BREAST

# 13

**Breast conditions** ........ 172

## ABBREVIATIONS

| | |
|---|---|
| **ALND** | Axillary lymph node dissection |
| ***BRCA*** | Breast cancer gene |
| **ER** | Oestrogen receptor |
| **FNA(C)** | Fine needle aspiration (cytology) |
| **HER2** | Human epidermal growth factor receptor 2 |
| **HRT** | Hormone replacement therapy |
| **LN** | Lymph node |
| **NACT** | Neoadjuvant chemotherapy |
| **NAET** | Neoadjuvant endocrine therapy |
| **NATT** | Neoadjuvant targeted therapy |
| **SLNB** | Sentinel lymph node biopsy |
| **SOB** | Shortness of breath |
| **TNM** | Tumour, nodes, metastases |

# Breast conditions

## NICE GUIDELINES ON REFERRAL

**Non-urgent referral:**

Patient <30y with unexplained breast symptoms

**Urgent 2ww:**

1. Patient ≥30y with unexplained breast/ axillary lump
2. Patient ≥50y with any unilateral nipple/ breast changes
3. Anyone with skin changes suspicious of breast cancer

**When to refer nipple discharge:**

1. Persistent, spontaneous unilateral discharge
2. Nipple inversion / eczematous skin changes

→ **Ix:** Examine for lump, if >35y refer for mammography

→ **Mx:** duct removal

**When to refer mastalgia for triple assessment:**

1. Pain not responsive to 3m of initial medical management
2. If any breast cancer red flags present (NICE guidelines above)

→ **Mx:** reassurance, NSAIDs/paracetamol, tamoxifen

**Paget's disease of nipple:**

→ rare spread of intraductal carcinoma

→ red, itchy, scaly areola

→ **DDx:** eczema

→ **Ix:** biopsy

**Risk factors for breast carcinoma:**

**Genetic (FHx):** *BRCA* +ve = 25% of risk

**Environmental:** = 75% of risk

- early menarche / late menopause
- nullip / late age of 1st child
- not breastfeeding
- HRT
- obesity, smoking

**Oestrogen exposure**

**Screening for all women 50–70y** (every 3y)

## Benign lumps

| | Fibroadenoma | Fibroadenosis / fibrocystic change | Cysts |
|---|---|---|---|
| **Pathogenesis** | **Benign** overgrowth of breast lobule | Localised fibrosis, inflammation & cyst formation | Fluid-filled lump that may associate with fibroadenosis |
| **Who?** | <30y<br>May grow in pregnancy | Between menarche & menopause<br>**Most common breast lump** | Perimenopause (35–50y) |
| **Presentation** | → firm, smooth, solid lump<br>→ **highly mobile** = 'breast mice'<br>→ usually painless | → lumpy breasts<br>→ may be mobile or fixed<br>→ **cyclical pain/swelling** | → rounded lump<br>→ **mobile**<br>→ occasionally painful |
| **Management** | 1. Reassurance<br>2. Surgical excision if large (>4cm)<br>1/3 regress, 1/3 stay same, 1/3 grow | 1. Reassurance<br>± Anti-inflammatories<br>± Hormone manipulation | 1. Reassurance<br>2. US-guided drainage (FNA)<br>3. Surgical removal if large |

Fat necrosis: similar presentation to fibroadenosis, but usually with history of trauma to the area (RF = obesity)

## Breast carcinoma

**CLASSIFICATION:** → can be **ductal** or **lobular**

1. **Non-invasive / *in situ* (5%)** = not breached basement membrane
2. **Invasive (85%)** = breached basement membrane

Invasive ductal carcinoma = most common (70%)

**CLINICAL PRESENTATION:**

- **Breast lump:** hard & irregular → fixed/tethered
- Palpable lymph nodes
- Nipple discharge / retraction ± Paget's disease rash
- Overlying skin changes – peau d'orange (dimpling)
- **Metastatic symptoms:** bone, lung, brain, liver, ovaries

**Prevalence & prognosis**

**Breast cancer = very common** (1 in 8 women)

15% breast cancers = HER2 +ve

70% breast cancers = ER +ve

**Best prognosis → Worst prognosis**

ER +ve → HER2 → Triple –ve

**INVESTIGATIONS: triple assessment**

1. **Clinical examination**
2. **Imaging** → <35y = USS (tissue too dense for mammography)
   → >35y = mammography (+USS if abnormal)
3. **Histology/cytology:**
   → FNAC if cystic
   → Core biopsy if solid (can grade tumour)

If USS of axilla shows lymph involvement then core biopsy of nodes

### TNM STAGING AND SURVIVAL[1]

| Tumour size | **T1** <2cm | **T2** 2–5cm | **T3** >5cm | **T4** extends to skin / chest wall |
|---|---|---|---|---|
| Nodes | **N0** no nodes | **N1** ipsilateral, axillary, mobile nodes | **N2** ipsilateral fixed axillary/mammary nodes | **N3** clavicular, axillary or mammary nodes |
| Metastases | **M0** no mets | **M1** mets | | |

| Stage 1 | Stage 2 | Stage 3 | Stage 4 |
|---|---|---|---|
| T1, N0, M0<br><2cm, no nodes | T1/2, N0/1, M0<br><5cm ± axillary nodes | T3, N1/N2, M0<br>>5cm + any nodes | M1<br>Any mets |
| **98%** 5y survival | **93%** 5y survival | **72%** 5y survival | **22%** 5y survival |

## Management of breast carcinoma[2]

### 1. NEOADJUVANT TREATMENT

- **NACT (neoadjuvant chemotherapy)** for any receptor status
- **NAET (neoadjuvant endocrine therapy)** if ER +ve
  e.g. tamoxifen, anastrozole
- **NATT (neoadjuvant targeted therapy)** if HER2 +ve
  e.g. Herceptin (trastuzumab)

### 2. PRIMARY TREATMENT

- **Surgery to the breast** – mastectomy or wide local excision ± oncoplastic/reconstruction procedures
- **Surgery to the axilla** – sentinel lymph node biopsy (SLNB) ± axillary lymph node dissection (ALND)
- **Primary endocrine therapy** if elderly / more frail

### 3. ADJUVANT TREATMENT → reduces recurrence

- Radiotherapy, chemotherapy, endocrine therapy, targeted therapy (see examples in neoadjuvant treatment)

### 4. TREATMENT OF METASTATIC DISEASE → depends on location of mets

- **If solitary node** = consider surgery
- **Symptomatic relief** = radiotherapy, chemotherapy, endocrine therapy

## Breast cancer recurrence

**Recurrence usually occurs in first 5y following treatment**

→ Needs reassessment & discussion with MDT to decide most appropriate management plan

**NB: risk of recurrence ↑s with size of tumour & LN involvement**

**Must closely monitor patients for symptoms of recurrence:**

- New lump in breast/axilla
- Constant arm/chest/shoulder pain
- Bone pain, weak/numbness, cough/SOB
- Nausea, anorexia, weight loss
- Headaches/confusion

**Management depends on:**

1. TNM stage (including size)
2. Location of lump
3. If lump is uni- or multifocal
4. HER2 / ER receptor status
5. Patient health & preference
6. Family history of breast cancer

**Indications for neoadjuvant treatment:**

- Reduce size of inoperable tumours
- Reduce size to allow breast conservation
- In inflammatory breast cancer
- Temporary contraindications to surgery
- Preparation to allow new drug testing

**SLNB** = dye injected into areolar area to identify drainage to sentinel node
→ *biopsy to assess spread*

**Inflammatory breast cancer**

= rare & aggressive subtype
**Sx:** sudden, red/swollen breast, peau d'orange
→ **no lump**
→ *early axillary spread*
**Mx:** neoadjuvant chemo, then surgery
***DDx:*** *infection*

---

[1] Cancer survival by stage at diagnosis for England, 2019. ONS.

[2] NICE (2018) *Early and locally advanced breast cancer* [NG101]

## Breast abscess & mastitis

### DEFINITIONS:

**Breast abscess:** walled-off collection of localised infection
**Mastitis:** inflammation of breast ± infection
**Duct ectasia:** periductal inflammation

### GENERAL SYMPTOMS:

- Warm, swollen, tender, erythematous breast/lump
- Milk stasis
- Nipple discharge (particularly in duct ectasia)
- ± Systemic infection / fever

### MANAGEMENT:

1. **Effective milk removal** – if lactating
2. **Empiric antibiotics**
3. **Analgesia** (paracetamol ± NSAID)

→ **If abscess:** aspiration & drainage (US-guided or in theatre)

**Risk factors for abscess/mastitis:**

- lactation / milk stasis
- skin infection
- underlying breast condition

Tight bra, difficulties breastfeeding

**Risk factors for duct ectasia**

- smokers around menopause

# VASCULAR DISEASE

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## ABBREVIATIONS

**aa.** – Arteries
**AAA** – Abdominal aortic aneurysm
**ABPI** – Ankle–brachial pressure index
**ABPM** – Ambulatory blood pressure monitoring
**ACEi** – Angiotensin-converting enzyme inhibitor
**ACS** – Abdominal compartment syndrome
**AF** – Atrial fibrillation
**AKI** – Acute kidney injury
**APS** – Antiphospholipid syndrome
**APTT** – Activated partial thromboplastin time
**ARB** – Angiotensin receptor blocker
**BB** – Beta-blocker
**CCB** – Calcium channel blocker
**CHF** – Congestive heart failure
**CKD** – Chronic kidney disease
**CLI** – Chronic limb ischaemia
**CLTI** – Critical limb-threatening ischaemia
**COCP** – Combined oral contraceptive pill
**COPD** – Chronic obstructive pulmonary disease
**CT CAP** – Computed tomography chest, abdomen & pelvis
**CTA** – Computed tomography angiography
**CTD** – Connective tissue disease
**CTPA** – Computed tomography pulmonary angiogram
**CVA** – Cerebrovascular accident
**CVD** – Cardiovascular disease
**DHP** – Dihydropyridine
**DM** – Diabetes mellitus
**DOAC** – Direct acting oral anticoagulant
**DVLA** – Driver and Vehicle Licensing Agency
**DVT** – Deep venous thrombosis
**EVAR** – Endovascular aneurysm repair
**HBPM** – Home blood pressure monitoring
**HDL** – High density lipoprotein
**HTN** – Hypertension
**IBD** – Inflammatory bowel disease
**ICA** – Internal carotid artery
**IMA** – Inferior mesenteric artery
**IVC** – Inferior vena cava
**IVDU** – Intravenous drug user
**JVP** – Jugular venous pressure
**LDL** – Low density lipoprotein
**LMWH** – Low molecular weight heparin
**LVH** – Left ventricular hypertrophy
**MI** – Myocardial infarction
**MSK** – Musculoskeletal
**OCP** – Oral contraceptive pill
**PAD** – Peripheral arterial disease
**PCKD** – Polycystic kidney disease
**PE** – Pulmonary oedema
**PSA** – Prostate-specific antigen
**PT** – Prothrombin time
**PTA** – Percutaneous transluminal angioplasty
**RA** – Rheumatoid arthritis
**RAAS** – Renin–angiotensin–aldosterone system
**RAS** – Renal artery stenosis
**R-F** – Radio-femoral
**RV** – Right ventricle
**SCA** – Subclavian artery
**SCV** – Subclavian vein
**SFJ** – Saphenofemoral junction
**SLE** – Systemic lupus erythematosus
**SMA** – Superior mesenteric artery
**SOB** – Shortness of breath
**TIA** – Transient ischaemic attack
**UO** – Urine output
**VTE** – Venous thromboembolism

# Hypertension

→ affects approx. 25% adults in UK
→ major cause of premature death

**Risk factors:**
- age >65y
- male
- FHx
- obese
- sedentary
- **salty diet**
- alcohol/caffeine
- DM
- renal disease

**Refer <40y with HTN for Ix of underlying cause:**

1. **RAS:** MRI, CT angiography, doppler
2. **CKD:** urinalysis, catecholamines, U&Es, eGFR
3. **Aortic coarctation:** R–F delay, MRI
4. **Endocrine cause:** renin, aldosterone, TFT

1. **Primary hypertension (95%)**
   - Cause unknown
2. **Secondary hypertension (5%) → suspect in younger patients**
   - Renal disease (80%) – glomerulonephritis, CKD, renal artery stenosis, PCKD
   - Endocrine – Conn's (↑ aldosterone), Cushing's (↑ cortisol), acromegaly (↑ growth hormone), hyperthyroidism
   - Drugs – steroids, NSAIDs, OCP, liquorice
   - Other – pregnancy, aortic coarctation, phaeochromocytoma

## Symptoms

**Essential HTN** (gradual ↑ BP over years) = **asymptomatic**

**Malignant HTN** (rapid, sustained ↑ BP) = **headaches, visual disturbances, renal dysfunction**

Diagnosed if SBP >200 **or** DBP >120 **and** bilateral retinal haemorrhages/exudates ± papilloedema

## Complications/consequences

- Heart – LVH → dilation & eventual failure
- Aorta – AAA & aortic dissection
- Brain – intracranial haemorrhage & stroke
- Kidney – CKD (glomerular destruction & nephron ischaemia)
- Eyes – hypertensive retinopathy

## Investigations

1. **Clinic BP** – 2 readings >140/90
2. **24h ABPM** (or HBPM) – **need 3× readings**
3. **Investigate target organ damage**
   - U&Es & urine dip (for proteinuria)
   - HbA1c, cholesterol, lipids
   - fundoscopy
   - ECG
4. **QRISK2** – to assess 10y risk of CVD

→ based on age, sex, ethnicity, BMI, DM, CKD, AF, FHx

**ABPM:** 2 readings per h for 1d (use average)
**HBPM:** 4 readings (2× am & 2× pm) for 7d (use average)

| | | |
|---|---|---|
| **Normal BP** | | <120/80 |
| **High BP** | | <140/90 |
| **Hypertension** | **Stage 1** | clinic BP >140/90 **and** ABPM >135/85 |
| | **Stage 2** | clinic BP >160/100 **and** ABPM >150/95 |
| | **Stage 3** | clinic systolic >180 **or** diastolic >110 |

## Management[1]

1. **Lifestyle:**
   - Diet: <1 tsp salt daily, fruit & veg, low fat dairy, low saturated fats
   - Exercise: >30min 4/5× a week
   - Weight loss: BMI 18–25
   - Smoking cessation & alcohol reduction: <14 units spread over week
2. **Medication if:**
   - Stage 1 if <80y **and** ≥1 of:
     - CVD / renal disease
     - end-organ damage
     - QRISK ≥20%
     - DM
   - All stage 2 or 3
3. **If treatment-resistant HTN** (with 3 medications) → refer for Ix of 2° cause

**Aims of Tx:**

<80y: BP <140/90
>80y: BP <150/90

**Follow-up: annual review**
- Of BP
- Of risk factors: smoking, alcohol, BMI, HbA1c
- Of medications & symptoms

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[1] NICE (2019, updated 2022) *Hypertension in adults* [NG136]

## PHARMACOLOGICAL TREATMENT:

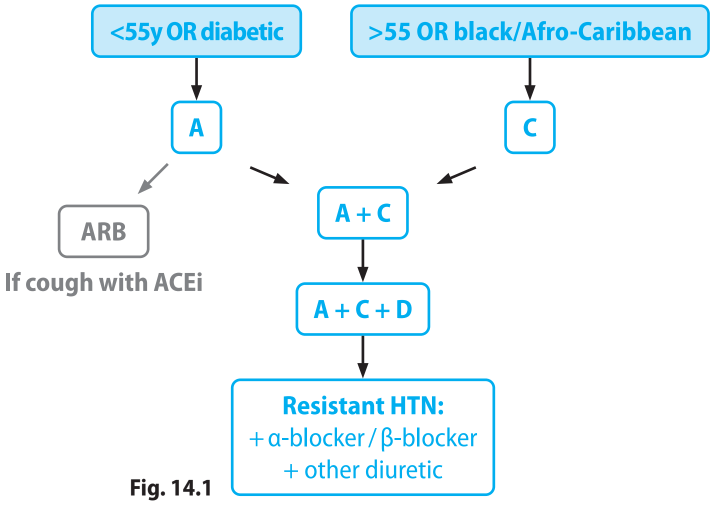

Fig. 14.1

**A = ACEi**, e.g. ramipril, lisinopril (start low & titrate up)
**ARB = angiotensin receptor blocker** e.g. losartan
**C = Ca channel blocker (DHP)** e.g. amlodipine, nifedipine
**D = diuretic (thiazide-like)** e.g. indapamide
**α-blocker** = doxazosin, prazosin
**β-blocker** = atenolol, bisoprolol
**Other diuretic** = low dose spironolactone (K⁺-sparing diuretic & aldosterone antagonist)

## Side-effects of treatment

### ACEis

| Side-effects: | Monitor: | Contraindications: |
|---|---|---|
| • cough<br>• hyperkalaemia<br>• angioedema<br>• ↓renal function<br>*nephroprotective (good for diabetics) | • U&Es<br>If eGFR drops by >20%, or Cr rises by 30%, **stop/lower dose** | • renovascular disease (GFR <30)<br>• caution + spironolactone<br>• pregnancy/breastfeeding |

### ARBs

| Side-effects: | Monitor: | Contraindications: |
|---|---|---|
| • hyperkalaemia<br>• angioedema<br>• ↓renal function | • U&Es | • renovascular disease<br>• pregnancy / breastfeeding |

### Spironolactone

| Side-effects: | Monitor: | Contraindications: |
|---|---|---|
| • hyperkalaemia<br>• gynaecomastia | • U&Es | • renovascular disease<br>• caution + ACEi |

| | |
|---|---|
| **α-blocker** | • postural hypotension |
| **β-blocker** | • bronchospasm (**avoid** in asthma) |

### Thiazide-like diuretics

| Side-effects: | Monitor: | Contraindications: |
|---|---|---|
| • hypokalaemia<br>• hyponatraemia / magnesium<br>• postural hypotension<br>• impaired glucose control<br>• exacerbate gout<br>*rely on renal excretion | • U&Es<br>• BP | • renal impairment<br>• gout<br>• DM<br>• pregnancy / breastfeeding |

### Calcium channel blockers

| Side-effects: | Monitor: | Contraindications: |
|---|---|---|
| • headache<br>• flushing<br>• ankle oedema<br>• hypotension<br>• gingival hyperplasia | • BP<br>• HR | • **avoid** rate limiting in CHF<br>• severe aortic stenosis |

# Carotid artery disease

→ results in **carotid artery stenosis**
→ most common cause = **atherosclerotic plaque** in cervical internal carotid artery

## Symptoms

- **Mostly asymptomatic**
- Can present with stroke / TIA / amaurosis fugax

## Investigations

- **Bloods:** FBC, WCC, CRP, cholesterol, lipids, HbA1c
- **Carotid arterial duplex USS**
- ± CT angiography

## Management[2,3]

### ASYMPTOMATIC STENOSIS

**<60%:** antiplatelet (aspirin/clopidogrel) ± statin
**60–99%:** antiplatelet ± statin ± carotid endarterectomy / stenting

→ Consider endarterectomy/stenting if >5y life expectancy, favourable anatomy, average surgical risk and radiological signs of 'high-risk for stroke' plaques

**100% (complete occlusion):** antiplatelet (aspirin/clopidogrel) ± statin

→ The main concern with carotid artery narrowing is ↑ velocity of flow, causing ↑ risk of emboli break-off & stroke. Total occlusion is therefore safer than high grade stenosis

### SYMPTOMATIC STENOSIS

**<50%:** antiplatelet (aspirin 300mg OD for 2w then 75mg clopidogrel for life) + statin

**≥50%:** dual antiplatelets + statin whilst awaiting further investigation

- **Carotid endarterectomy:** 1st line within 14d of event
- **Carotid stenting:** if high risk for anaesthesia or hostile neck (e.g. previous surgery/radiotherapy)

**100% (complete occlusion):** antiplatelet (aspirin 300mg OD for 2w then 75mg clopidogrel for life) + statin

**Grading severity of carotid artery disease:**

**Mild:** <50% stenosis
**Moderate:** 50–69% stenosis
**High grade:** 70–99% stenosis
**Complete occlusion:** 100% stenosis

**Risk factors for atherosclerosis:**

- DM
- Smoking
- Age
- Male
- HTN
- Obesity
- High cholesterol

**Complication:** STROKE
10–15% of all strokes are caused by embolisation from previously asymptomatic ICA stenosis >50%[2]

↓ Advise patients to inform DVLA & not drive for 1m post stroke/TIA

**Complications of carotid endarterectomy**

**Hypertension:** 40% (untreated can cause stroke)
**Stroke:** 1–3%
**Cranial nerve injury:** 1–3%

Switch to 75mg clopidogrel monotherapy after carotid endarterectomy or if no intervention planned

**HYPERLIPIDAEMIA:** ↑ LDL = ↑ cholesterol
**Primary: abnormal lipid metabolism:** familial genetic predisposition
**Secondary: metabolic disturbance:** obesity, DM, alcohol

**Investigations:**
**1. Hx & examination:** lifestyle RFs, BP, BMI
**2. Basic bloods:** FBC, LFT, U&Es, Cr, HbA1c
**3. Lipid levels:** total cholesterol, LDL, HDL

**Management:**
**1. Lifestyle factors**
**2. QRISK >10 indicates statin:** atorvastatin 20mg OD **at night**
(**consider fibrates** if statin contraindicated or in familial hyperlipidaemia)

**Serum cholesterol >4mM** predisposes to **atheroma** & ↑ mortality risk → calculate QRISK

**Target cholesterol** <200mg/dl
**Target LDL** <3.2 mmol/L (<2.6 if PAD or QRISK >20%)

**STATINS:** HMG-CoA reductase inhibitors
SEs: muscle ache, headache, nausea

---

[2] European Society for Vascular Surgery (2017) *Clinical Practice Guidelines: Management of Atherosclerotic Carotid and Vertebral Artery Disease*
[3] NICE (2019, updated 2022) *Stroke and transient ischaemic attack in over 16s* [NG128]

# Aneurysms

→ permanent localised dilatation with at least a 50% increase in vessel diameter

1. **True aneurysm:** involves **all layers** of artery wall
2. **False aneurysm:** abnormal dilation that **does not involve** all layers of arterial wall e.g. puncture in trauma/surgery

## Risk factors

- Smoking
- Age, male, FHx
- Hypertension
- CTDs

Diabetes = not a RF (may actually be protective)

## Abdominal aortic aneurysm (>3cm)

**SCREENING**[4,5] = one-off USS at 65y (1% prevalence)

| Size | Action |
|---|---|
| <3cm | **Normal:** no action |
| <4.5cm | **Small***: rescan in 12m |
| <5.5cm | **Med***: rescan in 3m |
| >5.5cm or ↑ by >1cm/y | **Large:** surgical repair |

→ 2ww referral

*Any newly diagnosed small or medium AAA should be seen within 12w in a vascular surgery clinic

**MANAGEMENT**[5,6]**:**

**Medical** (if <5.5cm): control BP, start statin + clopidogrel, **stop smoking**

Single, most important factor to reduce growth of AAA & improve fitness for surgery

**Surgical repair:** open or endovascular aneurysm repair (EVAR)

1. **Open surgery:** 7–10d in hospital + 3–6m recovery → *more durable repair (better for younger/fitter patients)*
2. **EVAR:** <2d in hospital + 2w recovery → *needs ongoing surveillance for leak (more suitable if older/frail)*

**AAA RUPTURE:**

**Signs of rupture:**

- Severe abdominal pain → **radiates to back/groin** ± **pulsatile mass**
- **Haemorrhagic shock:** hypotension, tachycardia, nausea
- **Unexplained collapse**

'Renal colic' in a man >55y = AAA rupture until proven otherwise

**Mx of rupture:** call vascular surgeon

1. ABCDE (X-match & **major haemorrhage protocol**)
2. Blood transfusion
3. FAST US scan (not diagnostic – quick test in ED)
4. CT angiogram (diagnostic)
5. Emergency surgery

**DDx of AAA rupture:**

- **Renal colic**
- Peptic ulcer perforation
- Pancreatitis
- Appendicitis
- Diverticulitis
- Mesenteric ischaemia

## Popliteal aneurysm (>1.5cm)[7]

**CLINICAL PRESENTATION:** often asymptomatic

- **Mass effect:** chronic limb ischaemia / gradual occlusion
- Thrombosis and distal embolisation: **acute limb ischaemia** (most feared complication)
- **DVT**
- **Rupture (rare):** acute limb ischaemia

50% have an AAA, 50% bilateral, 1/3 asymptomatic[7]

**SURGICAL REPAIR:** bypass graft **or** stenting

**Common sites of aneurysm:**

1. Abdo aorta (AAA)
2. Iliac aa.
3. Popliteal aa.
4. Femoral aa.
5. Thoracic aorta

**Complications of aneurysms:**

1. **Rupture:** especially aortic aneurysms
2. **Thromboembolism:** esp. popliteal → acute limb ischaemia
3. **Local compression:** back pain & venous congestion
4. **Fistulation:** aorto-enteric or aorto-caval

***Also operate if symptomatic****, no matter what size e.g. back pain, tender aneurysm, embolising*

**Prognosis of AAA rupture:** 50% mortality
**Prognosis of AAA repair:** <2% mortality

*Balance risks of surgery / patient fitness & recovery times with risk of rupture*

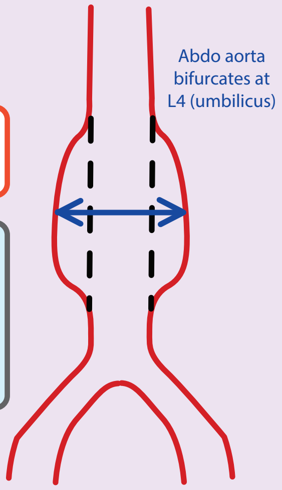

**Fig. 14.2** Diagramatic representation of an AAA.

**Indications for popliteal surgery:**

- >2cm
- Causing limb symptoms

---

[4] NHS Abdominal Aortic Aneurysm (AAA) Screening Programme

[5] NICE (2020) *Abdominal aortic aneurysm* [NG156]

[6] European Society for Vascular Surgery (2019) *Clinical practice guideline on management of abdominal aorto-iliac artery aneurysms*

[7] Diwan A, *et al.* (2000) Incidence of femoral and popliteal artery aneurysms in patients with abdominal aortic aneurysm. *J. Vasc. Surg.*, 31:863.

# Acute aortic syndromes

**Aortic dissection:** tear in the **intima** allowing blood to pass into the wall of the artery

**Intramural haematoma (IMH):** a contained haemorrhage into the aortic wall **without** a tear in the intima

**Penetrating aortic ulcer (PAU):** a lesion that ulcerates through the intima & beyond, potentially causing IMH or dissection

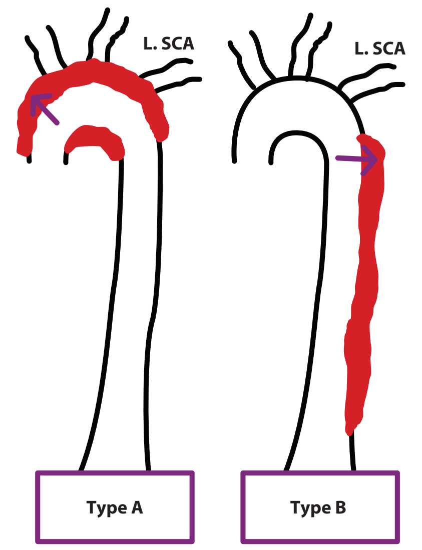

**Fig. 14.3** Types of dissection.

## Aortic dissection

| | Type A (70%) | Type B (30%) |
|---|---|---|
| **Location** | **Proximal** to left SCA<br>(ascending aorta) | **Distal** to left SCA<br>(descending aorta) |
| **Pulses** | Radio–radio delay | Radio–femoral delay |
| **Acute management** | **ABCDE** – $O_2$, fluids, X-match | |
| | **BP control + urgent surgery**<br>Aortic root replacement ± aortic valve replacement<br>30% mortality (1% per hour)<br>(BP control with IV beta-blocker) | **Control BP:** <120mmHg<br>(IV then PO antihypertensive)<br>+ monitored bed (e.g. on CCU)<br>*monitor UO, neuro obs, perfusion*<br>10% mortality |
| **Long-term management**[8] | **1.** Antihypertensives<br>**2.** Surveillance imaging (3-monthly, then 6-monthly, then yearly)<br>→ If type B have complications = surgical repair | |

**Indications for Type B repair:**
1. End organ malperfusion
2. Persistent pain
3. Refractory hypertension

## Complications

- **End organ damage:** as blood spreads distally & blocks aortic branches
  - **Coronary aa.:** MI
  - **Brachiocephalic trunk:** neuro signs / CVA
  - **Iliac aa.:** lower limb ischaemia
  - **Spinal cord ischaemia:** paraplegia
  - **Renal aa.:** AKI, haematuria
  - **SMA/IMA:** mesenteric ischaemia
- **Cardiac tamponade** = retrograde spread into pericardial sac
- **Aortic regurgitation**
- **Rupture:** through adventitia → massive haemorrhage

## Clinical presentation of aortic dissection

- **Severe, sudden tearing chest pain*** → radiates to back / down arm
- **Marked hypertension**
- **Signs of distal arterial trunk occlusion:** e.g. ↓ peripheral pulses
- **New murmur:** aortic regurgitation

*10% have no pain (especially likely if diabetic)

**Risk factors for dissection:**
- Atherosclerosis
- HTN
- CTD (Marfan/Ehlers–Danlos)
- Male

**Differentials:**
- **MI** ('crushing' + ECG changes)
- **PE** (dyspnoea + ABG changes)
- MSK back pain
- Pericarditis ('pleuritic' + ECG changes)

## Investigations

- **Bloods + ABG:** including X-match if suspect Type A or rupture
- **CXR:** widened mediastinum
- **CT aorta:** **diagnostic**
- **ECG:** exclude MI

CTA = gold standard for diagnosis ('double-barrelled' lumen seen)

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[8] European Society for Vascular Surgery (2015) *Clinical practice guideline on management of descending thoracic aorta diseases*

# Chronic peripheral arterial disease

## Causes

1. **Atherosclerosis:** DM, smoking, age, male, HTN, obesity, inactivity, cholesterol
2. **Vasculitis:** Buerger's disease → common in **young, heavy smokers**
3. **Fibromuscular dysplasia** → non-inflammatory arterial wall thickening
4. **Other:** cystic adventitial disease, iliac endofibrosis, popliteal artery entrapment

**Pathogenesis:** endothelial dysfunction → inflammation → macrophages → fatty streaks → plaque → rupture → platelet adherence → thrombus

## Signs and symptoms

| | Intermittent claudication | Ischaemic rest pain | Peripheral neuropathy<br>Often coexists alongside PAD in DM |
|---|---|---|---|
| **Pathology** | Insufficient perfusion **during exercise** | **Constant** insufficient perfusion | Damaged **peripheral nerves** |
| **Symptoms** | **Cramping muscle pain**<br>→ on exercise (limits walking*)<br>→ relieved by rest<br>→ reproduced walking **same distance** | **Continuous, severe, burning/aching**<br>→ worse at night<br>→ relieved by dangling leg out of bed<br>→ relieved by walking on a cold floor | **Tingling/numbness** ± pain<br>→ glove & stocking distribution<br>→ **no relief** dangling foot / cold floor<br>→ prone to wounds = infection risk |
| **Signs** | **Claudication distance* | • Absent/weak peripheral pulses<br>• Cold, pale, hairless legs<br>• Buerger's angle <20° (angle leg raised before pallor) | • Peripheral pulses present<br>• No pallor with Buerger's |

*Infection more likely if coexisting PAD*

## DDx of intermittent claudication

- **Spinal stenosis**
- **Venous claudication**
  - **bursting** pain from **start** of walking
  - relieved by **elevation**
- **Sciatica** = shooting pain
- **Popliteal artery entrapment**
  - normal pulses
  - younger, active, Sx when exercising

## Investigations

→ needs full cardiovascular screen

1. **History & examination:** peripheral vascular exam + BP, ECG
2. **Bloods:** FBC, lipids
3. **ABPI ± treadmill test**
4. **Duplex USS:** show site & degree of stenosis = non-invasive
5. **MR/CTA:** image larger aorto–iliac vessels = **invasive**

## Management[10]

Action is based on severity of symptoms

### MEDICAL

- **Lifestyle:** stop smoking, weight loss, exercise programmes (weekly for 3m)
- **Optimise comorbidities:** HTN, DM, cholesterol (high dose **statin**)
- **Antiplatelets:** 75mg clopidogrel (or 2.5mg rivaroxaban BD + 75mg aspirin)
- **Vasoactive drugs:** consider use in claudicants e.g. naftidrofuryl or cilostazol

**Fontaine classification:**

**Asymptomatic**
↓
**Intermittent claudication**
↓
**Ischaemic rest pain**
↓
**Ulceration/gangrene**

**Ischaemic rest pain** >2w + **ABPI <0.4 or TP <30mmHg** = **critical limb-threatening ischaemia (CLTI)** can cause development of **ulcers & gangrene** (↑ risk if DM) & needs **urgent Ix & Tx** to prevent limb loss

Leriche syndrome: occluded distal aorta, iliac & femoro-popliteal vessels
→ bilateral buttock/thigh pain ± erectile dysfunction

| Intermittent claudication | Spinal stenosis |
|---|---|
| Fixed claudication distance | Leg pain may be present at rest ± back pain |
| **Not** precipitated by ↑ time standing | Worsened by ↑ time standing |
| Worse walking uphill | Better walking uphill / bending forward |
| **O/E:** absent peripheral pulses | **O/E:** pulses present but **neurological** Sx |

$$\mathrm{ABPI}_{\mathrm{right}} = \frac{\text{right ankle pressure}}{\text{highest brachial pressure}}$$

| ABPI | Interpretation[9] | Action |
|---|---|---|
| >1.3 | Calcification (diabetes) | TBPI (toe BPI) |
| 1–1.3 | Normal | No action |
| 0.4–0.9 | **Mild–moderate PAD** (intermittent claudication) | **Routine** referral |
| <0.4 | **Severe PAD** (rest pain) | **Urgent** referral |
| <0.3 | **Impending gangrene** | |

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[9] Cronenwett JL & Johnston KW (2014) *Rutherford's Vascular Surgery*. 8th edition. Elsevier Health Sciences

[10] European Society for Vascular Surgery (2019) *Clinical practice guideline on management of chronic limb-threatening ischemia*

**SURGICAL → consider in more severe cases or where medical treatment has failed**
- Percutaneous transluminal angioplasty (PTA) or stenting
- Surgical reconstruction (bypass graft)
- Amputation = **last resort**

**Common sites of atherosclerosis**
- Coronary arteries
- Major branches of the aortic arch
- Visceral branches of abdo aorta
- Terminal abdominal aorta + branches

## Chronic mesenteric vascular occlusive disease

*→ atherosclerosis of **SMA, coeliac trunk & IMA****, *causing inadequate perfusion for digestion → 'gut claudication'*

**Good collateral blood supply so symptoms only likely to occur if ≥2 vessels affected*

**CLINICAL PRESENTATION:** (at least 3m duration)
- **Postprandial epigastric pain:** 10min to 3h after eating
- Gross weight loss & fear of eating (but appetite unaffected)
- Non-specific GI symptoms: altered bowel habits, N&V
- ± other vascular comorbidities e.g. HTN, DM, smoking etc.

**Risk factors for chronic mesenteric vascular occlusive disease:** as for all arterial disease (+ typically females >60y)

**INVESTIGATIONS: diagnosis of exclusion** (extensive GI examination needed to r/o other causes)
- History & examination: may have **epigastric bruit & tenderness**
- Routine bloods: often normal → may be nutritional deficiencies
- Duplex ultrasound of mesenteric vessels
- **CT angiography** → diagnostic

**Complications:**
- Malabsorption / weight loss
- Bowel infarction

**MANAGEMENT[11]: urgent if** significant weight loss, diarrhoea ± continuous pain

1. **Modify risk factors:** diet & exercise, stop smoking
2. **Antiplatelet** (75mg clopidogrel) **& high dose statin** (e.g. 80mg atorvastatin)
3. **Percutaneous mesenteric stenting:** less invasive than surgery
4. **Surgery:** bypass graft*

*SMA is the most important vessel to keep patent*

**If not suitable for endovascular intervention, endovascular intervention failed, or young with complex non-atherosclerotic lesions*

## Other causes of gut ischaemia

**ACUTE OCCLUSIVE MESENTERIC ISCHAEMIA:** due to embolism

**Sx:** severe, sudden abdominal pain

**RF:** AF, vascular disease

**Ix:** negative D-dimer can rule out → do not use lactate to diagnose or rule out

**Mx:** surgical embolectomy ± retrograde stenting; may need bowel resection

**Presentation of gut ischaemia:**

**Triad of Sx:** clinical diagnosis
1. Out of proportion pain
2. Diarrhoea &/or vomiting
3. Source of embolus e.g. AF

**NON-OCCLUSIVE MESENTERIC ISCHAEMIA (NOMI):** hypoperfusion causes ischaemia despite patent mesenteric vessels

**Sx:** severe abdominal pain

**Causes:** vasopressors, cocaine, abdominal compartment syndrome (ACS), dialysis, severe burns, cardiac surgery

**Mx:** surgical/endovascular revascularisation ± treatment of ACS with decompressive laparotomy

**ISCHAEMIC COLITIS:** acute, transient reduction in blood supply to large bowel

**Sx:** severe pain ± bloody stools

**Causes:** low BP / shock, thromboembolism, ↑ age, cocaine

**Mx:** supportive ± colectomy if peritonitic

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[11] European Society for Vascular Surgery (2017) *Clinical practice guideline on management of the diseases of mesenteric arteries and veins*

**MEDIAN ARCUATE LIGAMENT SYNDROME (MALS):** compression of coeliac arteries by diaphragm

**Sx:** chronic mesenteric ischaemia symptoms exacerbated in full expiration

**Mx:** surgical release of median arcuate ligament

**VENOUS MESENTERIC ISCHAEMIA:** due to venous thrombosis in the SMV ± extension into splenic vein or portal vein

**Sx:** generalised abdominal pain, nausea ± haematemesis ± melaena ± haematochezia

**RFs:** abdominal malignancies, myeloproliferative disorders, thrombophilia, pancreatitis, IBD

**Mx:** NG tube, fluid, analgesia **and** anticoagulation (LMWH eventually converted to DOAC/warfarin)

± surgical bowel resection (if ischaemic bowel)

## Renal artery stenosis

*→ results in ↓ renal perfusion → RAAS activation → **hypertension***

### CAUSES:

→ **90% atherosclerosis**

→ 10% fibromuscular hyperplasia (young males)

### CLINICAL PRESENTATION:

- **Resistant hypertension** (RAAS activation)
- Worsening renal function (especially ACEi-induced that results in flash pulmonary oedema)

### INVESTIGATIONS: rule out Conn's & phaeochromocytoma

1. **Hx & examination:** BP, urine dip
2. **Bloods:** especially U&Es
3. **Renal USS**
4. **CT/MRI** – 'string-of-beads' appearance if fibromuscular
5. **Renal angiography** = gold standard

### MANAGEMENT:

Monitor closely if bilateral stenosis

1. **Antihypertensive therapy:** ACEis/ARBs → **can cause large ↑ in creatinine** (measure before & after)
2. **Statin + antiplatelet**
3. **Surgery:** PTA or aorto–renal endarterectomy/bypass → only if severe disease and not if artery occluded

# Acute limb ischaemia

= **sudden decrease in arterial limb perfusion** (due to thrombus/embolism)

1. From **acute exacerbation of CLI**
2. From **arterial thromboembolism**
3. From **iatrogenic cause**

## Causes

| | Embolism | Thrombosis |
|---|---|---|
| **Onset** | **Sudden** | **Slower** |
| **Severity** | **Very:** no collaterals | **Less:** advanced collaterals |
| **Pulses** | Present in contralateral limb | Long-term **bilateral ↓ pulses** |
| **History** | No PHx ischaemic Sx | PHx ischaemic Sx |
| **Aetiology** | **Cardiac**<br>• AF (80% of embolic causes)<br>• Mural thrombus secondary to MI<br>• Endocarditis<br>• Paradoxical<br>• Atrial myxoma<br>**Non-cardiac**<br>• Atheroembolism (from plaques)<br>• Aortic mural thrombus | • Atherosclerotic obstruction (ruptured plaque, popliteal aneurysm)<br>• Occluded graft / endovascular stent<br>• Hypercoagulable states (e.g. cancer, Covid-19, thrombophilia)<br>• Aortic dissection<br>• Illicit IV drug use |

**Symptoms: 6 Ps**

**P**ain – constant
**P**allor ± cyanosis
**P**ulseless – especially peripheral
**P**aralysis – nerve ischaemia
**P**araesthesia – ↓ sensation
**P**erishingly cold

**Irreversible ischaemia** = fixed mottling & blistering **and** hard, woody muscles
**→ needs amputation**

## Classification of acute limb ischaemia

**Rutherford classification**

| Category | Description | Capillary return | Muscle paralysis | Sensory loss |
|---|---|---|---|---|
| **I. Viable** | No immediate threat | Intact | None | None |
| **IIa. Threatened** | Salvageable if prompt Tx | Intact/slow | None | Partial |
| **IIb. Threatened** | Salvageable if immediate Tx | Slow/absent | Partial | Partial/complete |
| **III. Irreversible** | Amputation | Absent | Complete | Complete |

## Investigations

1. Examination
2. ABPI
3. Bloods (FBC, U&Es, coag, G&S, ABG)
4. Arterial duplex ± CT angiogram
5. ECG

**Differentials:**

1. **Chronic peripheral neuropathy** – limb warm & pulses present
2. **Compartment syndrome** – pain on passive flexion/extension
3. **DVT** – red, hot, swollen & pulses present

## Management[12]

untreated = limb loss &/or death

1. **Urgent admission:** only **6h** for revascularisation **→ surgical emergency**
2. **IV unfractionated heparin:** prevents clot propagation (stat bolus followed by continuous IV infusion → measure APTT 4–6h after commencing)
3. **Surgical revascularisation:**
   - **Embolus:** surgical embolectomy (clot retrieval)
   - **Thrombus:** surgical bypass or endovascular therapy e.g. suction thrombectomy, mechanical atherectomy
   - **Consider catheter-directed thrombolysis** (alteplase) – **if Rutherford IIa/b, no contraindications, on an individual basis**
4. **Find & treat underlying cause**
   - CT aorta – image **entire aorta** to look for proximal causes of embolisation
   - Echocardiogram
   - Tumour markers
   - Thrombophilia screen

Long-term anticoagulation (e.g. warfarin or DOAC)

**Reperfusion injury:** major cause of mortality

→ Sudden return of acidotic blood causes **systemic inflammatory response**, oxidative damage & capillary leakage
→ High concentration of **potassium & anaerobic metabolites** → can lead to arrhythmia and cardiac arrest
→ **Myoglobinuria** ('Coca-Cola' urine) may cause worsening of renal function
→ Can cause **compartment syndrome*** = ***out of proportion pain*** *and on* ***passive stretch***
(see *Chapter 19: Trauma & orthopaedics*)

*consider calf fasciotomies at time of revascularisation

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[12] European Society for Vascular Surgery (2020) *Clinical practice guidelines on the management of acute limb ischaemia*

# Amputation

→ 80% due to peripheral arterial disease

## Indications

- Relieve ischaemic rest pain (where surgical Tx ineffective)
- Limb condition threatening life (gas gangrene / severe infection)
- Trauma (causing non-functionable limb)

## Level of amputation

→ *through **healthy tissue,** with **good blood supply** & preferably at **site suitable for a prosthesis***

**MINOR AMPUTATION:** below ankle

→ Most often digital (toe) or trans-metatarsal amputation secondary to diabetic foot sepsis

→ Aim to establish arterial flow into foot to allow resultant wound to heal

**MAJOR AMPUTATION:** above ankle → if more extensive ischaemic insult

- **Below knee (trans-tibial)*** = greater functional success than above knee but requires well-perfused calf
- **Above knee (trans-femoral)**
- **Through knee amputation**
- **Hip disarticulation**
- **Hind-quarter amputation**

*not if fixed flexion deformity of knee or severe OA

## Rehabilitation

→ *aim is for patient to function independently in ADLs*

**PRE-OPERATIVE:**

- Pre-amputation consultation with physiotherapist and occupational therapist
- Establish if patient will be able to use prosthesis
- Is home suitable for wheelchair use? (all patients are discharged in a wheelchair)

**EARLY POST-OPERATIVE:**

- Initial exercises to prevent knee flexion deformity and muscle strengthening exercises
- Practise transferring and wheelchair mobility
- Consider use of pneumatic post-amputation mobility (PPAM) aid from 1w

**LATE POST-OPERATIVE:**

- Once wound has healed, prosthetic measurements commence at 6–8w
  - ↳ **Below-knee amputees** = 50% achieve community ambulation with prosthetic
  - ↳ **Above-knee amputees** = 26% achieve community ambulation with prosthetic

## Complications of amputation

| Early | Late |
|---|---|
| • Wound dehiscence<br>• Infection | • Contractures<br>• Phantom limb pain |

**Outcome:**

- 30d mortality 15%
- 5y mortality 65%

50% of diabetics with major limb amputation will have contralateral leg amputated in 2–3y

It requires **15% more energy** to walk with a below-knee prosthesis & **>20% more energy** with an above-knee prosthesis

# Vasospastic disorders

## Pathophysiology

Arterial spasm → reduced perfusion → ischaemia → necrosis

## Types

**Acrocyanosis** = **benign vasospasm of cutaneous arteries**
→ **Persistent** diffuse cyanosis of hands & feet

**Livedo reticularis** = **often secondary to an underlying condition**
→ **Persistent mottled** cyanosis of skin e.g. APS, SLE, polycythaemia

**Raynaud's syndrome** = **paroxysmal vasospasm of peripheral arterioles**
→ **Intermittent** cyanosis of hands/feet and digits

Raynaud's syndrome affects 5% of the population

## Raynaud's syndrome

**Raynaud's disease:** genetic component (young females)

**Raynaud's phenomenon:** secondary to another condition

**Must screen for these O/E & with blood tests**

- **Autoimmune:** scleroderma, SLE, RA, Sjögren's, dermatomyositis
- **Arterial disease:** atherosclerosis, thoracic outlet obstruction, Buerger's disease
- **Endocrine:** DM, hypothyroidism
- **Blood:** lymphoma, polycythaemia
- **Environmental:** smoking, trauma, chemicals, chronic vibration
- **Drugs:** beta-blockers, COCP, cytotoxic agents

**Features suggesting 2° disease:**

- presentation in early childhood **or** >30y
- asymmetrical distribution
- male

**Buerger's disease** (thromboangiitis obliterans) = peripheral ischaemia associated with **smoking**

**CLINICAL PRESENTATION:** colour changes / numbness in digits + **pain/burning on rewarming**

PALE (ischaemia) → BLUE (cyanosis) → RED (reactive hyperaemia)

**Triggers:**
→ **cold**
→ emotion
→ smoking
→ exercise

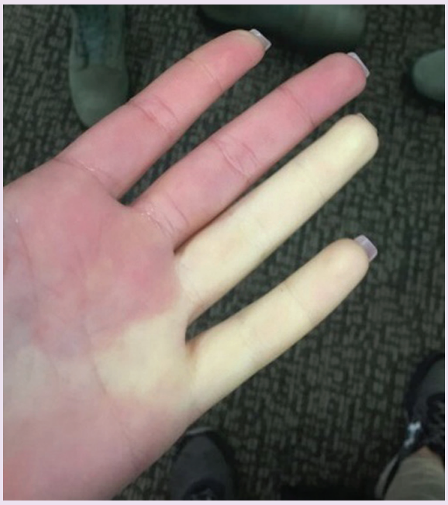

**Fig. 14.4** Raynaud's syndrome.

**INVESTIGATIONS:**

1. **Hx & examination**
2. **Bloods:** FBC, U&Es, LFT, TFT, coag, glucose
3. **Autoimmune screen:** ANA, CRP

**MANAGEMENT:**

Refer to rheumatology / other specialty if suspect underlying cause

**Mild/moderate disease:**
→ **Lifestyle:** stop smoking, warm clothing / avoid cold & other triggers (e.g. COCP/BBs)
→ ± **Vasodilators:** CCBs (nifedipine)

**Severe disease:**
→ **IV Prostaglandins:** iloprost
→ ± **Surgery** = last resort (sympathectomy, embolectomy)

# Thoracic outlet syndrome

**→ compression of neurovascular bundle within thoracic outlet**

**SCV, SCA & brachial plexus**

## Causes

- Cervical rib
- Healed clavicular fracture
- Scalene muscle hypertrophy
- Malignancy (Pancoast tumour)

## Clinical presentation

- **Venous:** DVT & peripheral swelling
- **Arterial:** claudication / limb ischaemia
- **Neuro:** pain, paraesthesia, weakness, muscle wasting → T1 distribution (inner arm)

## Investigations

1. **Hx & examination**
2. **Bloods:** FBC, clotting
3. **CXR** – show cervical ribs
4. **Duplex USS** – dynamic vascular compression
5. **Nerve conduction studies**
6. **MRI** – with arms fully abducted

## Management

**Venous:** thrombolysis ± venoplasty / surgical decompression
**Arterial:** surgical decompression ± angioplasty/bypass
**Neurological:** physio & Botox ± surgical decompression

PLUS removal of cervical rib if present, or first rib resection + scalenectomy

**DDx:** cervical myelopathy

# Varicose veins

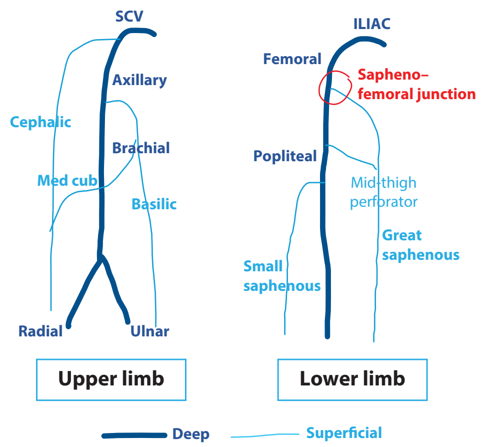

**Fig. 14.5** Venous systems of the upper & lower limbs.

**Thrombophlebitis without suspicion of DVT is treated with topical NSAIDs only*

### Classifying symptoms:

- **CEAP classification:** Clinical, aEtiological, Anatomical, Pathophysiological
- **Venous Clinical Severity Score** (VCSS)

### Indications for NHS Tx:

→ Recurrent thrombophlebitis
→ Venous ulcers
→ Bleeding
→ Chronic skin changes
(eczema/lipodermatosclerosis)

### Signs of chronic venous insufficiency:

**Varicose veins**
**Venous ulcers**
**Venous stars (spider naevi)**
**Lipodermatosclerosis**
**Atrophie blanche**
**Pigmentation / pitting oedema**
**Scars**

## Pathophysiology of venous disease

### Normal physiology:

- **Venous return driven by:**
  1. Muscle contraction
  2. ↓Intrathoracic pressure with inspiration
- **Blood flow** = superficial → perforating → deep veins
- **Valves prevent BACKFLOW** (absent in vena cava & common iliacs)

### Venous disease results from VALVE INCOMPETENCE:

- **In deep veins** = deep venous insufficiency (usually after DVT)
- **In superficial veins** = VARICOSE VEINS

## Definition

**Varicose veins** = tortuous, dilated veins
→ **Primary:** idiopathic or no valve defect (women, pregnancy)
→ **Secondary:** to deep venous insufficiency (DVT or ↑ pelvic pressure)

***Post-thrombotic syndrome (PTS):*** *chronic venous insufficiency 2° to DVT*

## Clinical presentation

- Cosmetic
- May be itchy (venous eczema)
- May cause heaviness/aching & ankle oedema
- Venous ulceration

## Complications

### ACUTE

- **Thrombophlebitis:** inflamed vein due to clot → red, painful, hot
  **Tx:** topical NSAIDs* **or** LMWH (if >5cm and within 3–5cm of SFJ)
- **Bleeding:** due to skin thinning (most often after a hot shower)
  **Immediate Tx:** elevation + compression

### CHRONIC VENOUS INSUFFICIENCY

- **Venous ulcers:** 50% caused by varicose veins
- **Venous eczema:** red & flaky skin
- **Lipodermatosclerosis:** brown haemosiderin 'champagne bottle'

## Diagnosis

- **Duplex USS**

## Management[13]

1. **Lifestyle:** weight loss, exercise, avoid prolonged standing
2. **Compression stockings:** must r/o PAD first with ABPI (>0.8)
3. **Endothermal catheter ablation:** radiofrequency catheter passed up great saphenous vein to heat vein & seal it
4. **Foam sclerotherapy:** inject foam containing sclerosing agent into vein to seal vein
5. **Surgery:** disconnect great saphenous vein and strip it out

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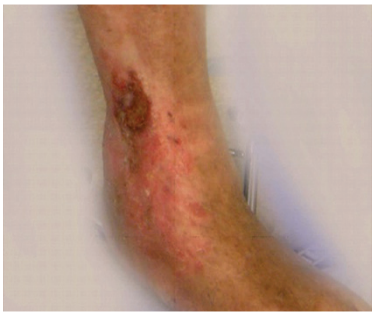
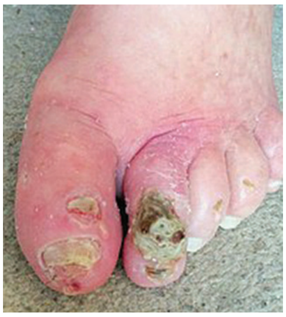
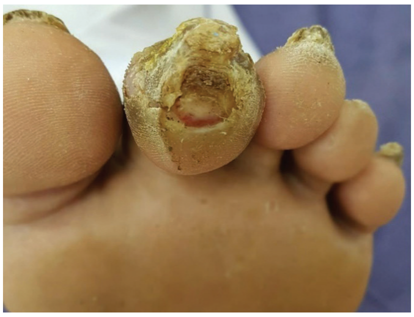
[13] NICE (2013) *Varicose veins* [CG168]

# Leg ulcers

| | Venous (70%) | Arterial | Neuropathic |
|---|---|---|---|
| **Pathophysiology** | • Valve incompetence & reflux<br>• Calf muscle dysfunction<br>= toxins accumulate → inflammation and necrosis | • Atheromatous changes = compromised blood flow<br>= hypoxia & toxins accumulate → inflammation and necrosis | • Peripheral neuropathy<br>= loss of protective sensation – trauma goes unnoticed<br>• Vascular disease & infection<br>= reduced wound healing |
| **Risk factors** | DVT, varicose veins, age, pregnancy, surgery | Diabetes, HTN, smoking, arterial disease, cholesterol emboli, Raynaud's disease, trauma | Diabetes, trauma, prolonged pressure, alcohol excess, B12/folate deficiency |
| **Location** | Gaiter area<br>(between malleolus and knee) | Bony prominences<br>(lateral malleolus and toes) | Pressure areas<br>(plantar aspect of foot, toes) |
| **Features** | • Large and irregular<br>• Shallow with sloping edges<br>• Granulation tissue<br>= may be painful | • Smaller and round<br>• 'Punched-out' borders<br>• Little granulation tissue & dry<br>= very painful | • Small, round, deep<br>• 'Punched-out' borders<br>• Thick rim<br>= painless |
| **Leg condition** | • Lipodermatosclerosis<br>• Venous eczema<br>• Haemosiderin (red/brown)<br>• Atrophie blanche (smooth white sclerotic plaques)<br>• Heavy, aching, pruritus, oedema | • Claudication / ischaemic rest pain symptoms<br>• Cool, hairless, dry, shiny skin | • Surrounding callus<br>• Loss of sensation<br>• Dry, cracked skin<br>**High risk of developing diabetic foot sepsis** |
| | <br>Fig. 14.6 | <br>Fig. 14.7 | <br>Fig. 14.8 |
| **Specific treatment** | • Elevation and compression<br>**1st-line:** 4-layer bandaging<br>**Other:** stockings<br>• Superficial venous surgery (prevents recurrence and accelerates healing)<br>**Must exclude coexisting arterial insufficiency before starting compression therapy (with ABPI)** | • Manage vascular risk factors<br>e.g. antiplatelets, stop smoking<br>• Surgical revascularisation (to restore vascular flow) | • Optimise glycaemic control<br>• Treat coexisting arterial disease<br>• Good foot care<br>• X-ray/MRI to find osteomyelitis<br>• Offload pressure (therapeutic footwear) |
| **General treatment** | • Dressings ± antibiotics ± emollients<br>• Debridement: dressings, surgery, larvae | | |

# Venous thromboembolic disease

**All patients have VTE risk assessment on admission**
→ prophylactic LMWH if indicated

**Differentials of DVT:**
→ **Cellulitis:** ± rash/pus
→ Chronic venous insufficiency: chronic skin change signs
→ Superficial thrombophlebitis: erythema/inflammation along vein
→ Acute arterial ischaemia: pallor, pain, pulseless, etc.
→ Baker's cyst: swelling behind knee & knee stiffness

**Wells score for DVT:**

| Clinical features | Points |
|---|---|
| Active cancer (including current Tx or Tx in past 6m) | 1 |
| Paralysis / paresis / plaster immobilisation of lower limb | 1 |
| Bedridden ≥3d or major surgery in past 12w | 1 |
| Localised tenderness along deep venous system | 1 |
| Entire leg swollen | 1 |
| Calf swelling ≥3cm larger than other leg | 1 |
| Pitting oedema confined to symptomatic leg | 1 |
| Collateral superficial veins (non-varicose) | 1 |
| Previous DVT | 1 |
| Alternative diagnosis at least as likely as DVT | −2 |

If suspicion of DVT/PE, **start treatment dose LMWH** while waiting for scan results to confirm or r/o

**Differentials of PE:**
→ MI/angina
→ Pneumonia
→ CHF
→ COPD/asthma exacerbation
→ Pneumothorax
→ Cardiac tamponade

## Risk factors

**Virchow's triad:**

1. **Blood stasis:** immobility, surgery, varicose veins, long-haul travel
2. **Hypercoagulability:** obesity, pregnancy, dehydration, OCP, thrombophilia, infection, malignancy, IVDU
3. **Endothelial damage:** age, trauma/surgery (especially orthopaedic)

(**PLUS:** PHx or FHx of VTE 1st-degree relative)

## Deep vein thrombosis (DVT)

→ *thrombus in one of the deep veins, commonly in calves or thighs*

### CLINICAL PRESENTATION:

**Unilateral symptoms:**

- Leg tenderness, warmth, swelling, erythema
- Pitting oedema
- Distended superficial veins / thrombophlebitis
- **Homans sign:** pain on dorsiflexion of foot

### INVESTIGATIONS:

(Hx & examination)

1. **Wells score:** determines next step
2. **D-dimer:** not specific for DVT/PE
3. **Compression USS:** if no compression = clot
4. **Duplex USS:** shows clot (**diagnostic**)

### INITIAL MANAGEMENT[14]:

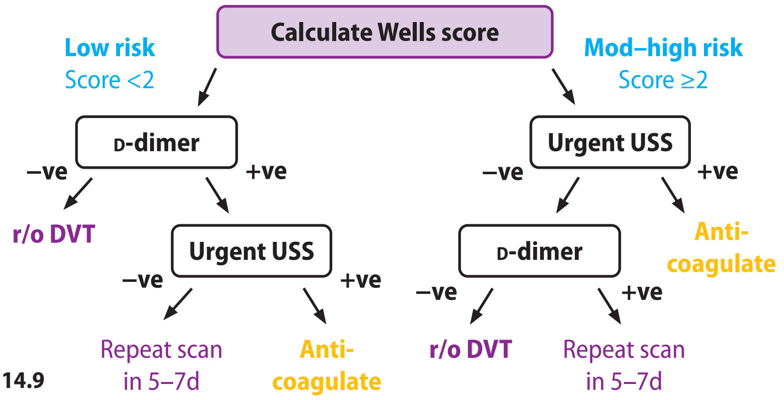


**Fig. 14.9**

## Pulmonary embolism (PE)

→ *clot in pulmonary artery (usually embolism from DVT)*

### CLINICAL PRESENTATION: sudden onset

- Acute onset SOB
- Pleuritic pain
- Haemoptysis

O/E: tachycardia, hypotension, low-grade fever, ↑ JVP, signs of DVT

### INVESTIGATIONS:

1. **Wells score:** determines next step
   → **<4** = D-dimer (if +ve = urgent CTPA)
   → **>4** = Urgent CTPA (V/Q scan if CTPA contraindicated)
2. **Bloods:** FBC, LFTs, U&Es, clotting, ABG
3. **CXR:** usually normal
4. **ECG:** sinus tachycardia (S1Q3T3)
5. **Echo:** may be considered to look for right heart strain

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[14] NICE (2020) *Venous thromboembolic diseases* [NG158]

**Categorisation of PE:**

1. **Massive (5%):** >50% pulmonary circulation blocked
   → chest pain, severe SOB, **hypotension** & hypoxia
2. **Major (10%):** mid-sized pulm arteries blocked
   → chest pain, SOB, haemoptysis
   **(ECG = RV strain)**
3. **Minor (85%):** small peripheral vessels blocked
   → may be asymptomatic, SOB
   **(no ECG changes)**
4. **Subacute:** multiple small/medium emboli accumulate
   → **over weeks** = ↑ JVP, cor pulmonale

## Management of thromboembolic disease[15]

1. **ABCDE:** $O_2$, fluids, analgesia
2. **ANTICOAGULATION:**
   - **Acute:** IV LMWH initially and then switch to ongoing therapy:
     - **1st-line:** DOAC (apixaban/rivaroxaban)
     - **Alternative:** LMWH (until INR in range) then warfarin
   - **Ongoing:** Continue anticoagulation for **3m**
     (if unprovoked event / high risk = continue **6m**)

NB: thrombolysis (alteplase) only indicated if *massive PE* / resistant to fluid resuscitation / at risk of cardiac arrest

3. **INVESTIGATE FOR CAUSE:** only if not obvious / unprovoked **OR** were abnormalities on examination / routine bloods
   - **Bloods:** FBC, U&Es, LFT, clotting, coag screen (APTT/PT)
   - **Antiphospholipid antibodies:** APS
   - **Cancer screen:** bloods, urinalysis, CXR, PSA/mammogram
   - **CT scan:** may show iliac artery compression

NB: if features suggesting cancer = immediate CT CAP (if not do bloods first, then CT CAP if concern of cancer)

4. **If MASSIVE or PROXIMAL DVT (extending above common femoral vein):**
   - CT venogram + referral to vascular surgery
   - Consideration of catheter-directed thrombolysis ± venous stenting

**If high risk of PE & CTPA not immediately available, then start anticoagulation**
(don't wait for CTPA confirmation)

**If anticoagulation is contraindicated, or recurrent VTE despite anticoagulation, consider an IVC filter**

**Antiphospholipid syndrome (APS):**
Diagnosed if antibodies present for ≥12w + unexplained thrombotic event

Compression of left iliac artery = May–Thurner syndrome

---

[15] NICE (2020) *Venous thromboembolic diseases* [NG158]

# Lymphoedema

**Chronic swelling** due to ↑ fluid in soft tissues as the result of **failed lymphatic drainage**

## Aetiology

**PRIMARY:** due to inherited abnormality of lymphatic vessels (e.g. Milroy's disease)
→ presents early in life

**SECONDARY:** due to obstruction of / damage to lymphatic vessels
→ infection/cellulitis
→ venous disease / DVT
→ immobility/obesity
→ trauma
→ liver disease
→ heart failure
→ malignancy
→ renal disease
→ cancer surgery / radiotherapy

## Clinical presentation

- **Non-pitting oedema** usually in legs & arms

## Investigations

***Aim:*** *r/o serious underlying condition e.g. CHF, malignancy, renal disease*

1. **Careful Hx & examination** → measure circumference of limb
2. **Lymphoscintigraphy** → radioactive dye injected into lymphatic system

## Management

**Decongestive lymphatic therapy (DLT):**

1. Compression bandages
2. Skin care ± ABX – reduce risk of cellulitis
3. Exercise/mobilisation
4. Specialised massage techniques

# 15

# UROLOGY

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## ABBREVIATIONS

**#** – Fracture
**AAA** – Abdominal aortic aneurysm
**AFP** – Alpha-fetoprotein
**AKI** – Acute kidney injury
**ALP** – Alkaline phosphatase
**BCG** – Bacille Calmette–Guérin (TB vaccine)
**BPH** – Benign prostatic hyperplasia
**CF** – Cystic fibrosis
**CKD** – Chronic kidney disease
**Cr** – Creatinine
**CT KUB** – CT kidneys, ureters, bladder
**DM** – Diabetes mellitus
**DRE** – Digital rectal examination
**ESWL** – Extra-corporeal shock wave lithotripsy
**FSH** – Follicle-stimulating hormone
**hCG** – Human chorionic gonadotrophin
**HPCR** – High pressure chronic retention
**IAP** – Intra-abdominal pressure
**IPSS** – International Prostate Symptom Score
**LDH** – Lactate dehydrogenase
**LH** – Luteinising hormone
**LUTS** – Lower urinary tract symptoms
**MSK** – Musculoskeletal
**MSU** – Mid-stream urine
**PCKD** – Polycystic kidney disease
**PCNL** – Percutaneous nephrolithotomy
**PSA** – Prostate-specific antigen
**PTH** – Parathyroid hormone
**PTH-rp** – Parathyroid hormone-related protein
**PUJ** – Pelvic–ureteric junction
**RTA** – Road traffic accident
**SCD** – Sickle cell disease
**STI** – Sexually transmitted infection
**TB** – Tuberculosis
**TURBT** – Transurethral resection of bladder tumour
**TURP** – Transurethral resection of prostate
**UTI** – Urinary tract infection
**VHL** – Von Hippel–Lindau disease
**VUJ** – Vesico-ureteric junction

# Stone disease (nephrolithiasis)

**Complications:**
Pain, infection, sepsis, hydronephrosis & post-renal AKI, obstructive uropathy

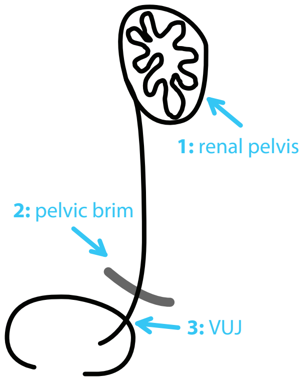


**Fig. 15.1** Common sites of obstruction.

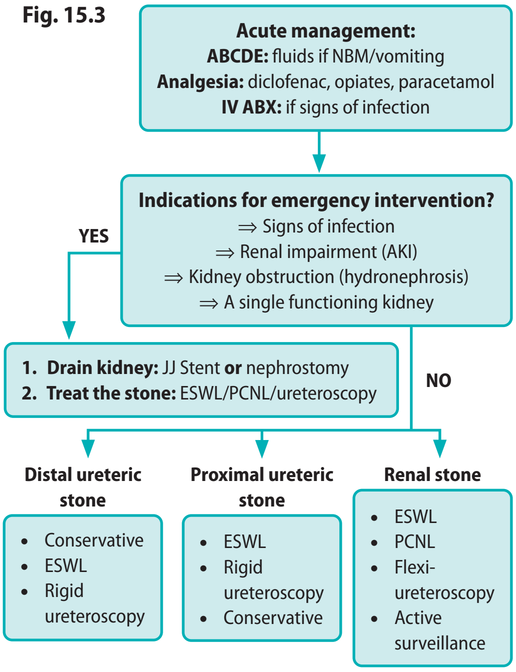
**Common sites of obstruction = narrowings:**

1. Renal pelvic–ureteric junction (PUJ)
2. Pelvic brim
3. Vesico-ureteric junction (VUJ)

**Differentials:**

- Gynaecology (e.g. ectopic, ovarian cyst etc.)
- Pyelonephritis
- Appendicitis
- MSK pain
- Bowel obstruction
- Ruptured AAA
- Biliary pathology

**Fig. 15.3**



**Acute management:**
**ABCDE:** fluids if NBM/vomiting
**Analgesia:** diclofenac, opiates, paracetamol
**IV ABX:** if signs of infection

**Indications for emergency intervention?**
⇒ Signs of infection
⇒ Renal impairment (AKI)
⇒ Kidney obstruction (hydronephrosis)
⇒ A single functioning kidney

YES

1. **Drain kidney:** JJ Stent **or** nephrostomy
2. **Treat the stone:** ESWL/PCNL/ureteroscopy

NO

**Distal ureteric stone**
- Conservative
- ESWL
- Rigid ureteroscopy

**Proximal ureteric stone**
- ESWL
- Rigid ureteroscopy
- Conservative

**Renal stone**
- ESWL
- PCNL
- Flexi-ureteroscopy
- Active surveillance

## Clinical presentation

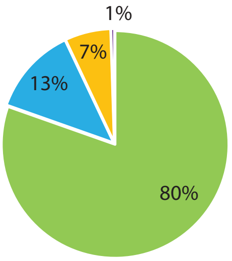
**Acute:** Loin → groin pain, flank tenderness, palpable kidney, infection, haematuria
**Chronic:** renal failure (oligo-/anuria)

Often restless/cannot keep still

## Risk factors

- **Dehydration**
- Diet (high protein, high salt)
- FHx or PHx
- Repeated UTIs
- Endocrine disorders (Addison's, Cushing's, hyperparathyroidism, hypercalciuria, cystinuria, gout)
- Anatomical abnormalities (horseshoe kidney, strictures, PUJ obstruction)
- Drugs (steroids, chemotherapy, loop diuretics)

## Types of stone[1]



🟩 Calcium → **40%** Calcium oxalate, **4%** Calcium phosphate, **36%** Mixture } (metabolic)

🟦 Struvite → Magnesium, ammonium, phosphate (UTIs)
→ 'Staghorn calculi'

🟨 Uric acid → High levels of blood purines (diet/haematological)
→ Radiolucent

🟪 Cystine → Familial disorders affecting cystine transport in kidneys
→ Smell like sulphur

**Fig. 15.2**

## Clinical presentation

1. **URETERIC COLIC:** sudden onset loin → groin pain (in waves)
2. **Nausea & vomiting**
3. **Haematuria** (often microscopic)
4. **Concurrent infection:** fever, rigors, sepsis

cramping pain that comes in waves

## Investigations

1. **Hx & examination:** renal angle tenderness
2. **Urine dip & MCS** – for infection, urine pH & haematuria
3. **Bloods:**
   - FBC, CRP/ESR, clotting, VBG (metabolic acidosis)
   - U&Es (↑ Cr suggests AKI/dehydration)
   - Ca, $PO_4$, urate, glucose
4. **Basic imaging: non-contrast** CT KUB = **gold standard** for stone visualisation
5. **Specialist imaging:** CT urogram (only if urology request)

*** PREGNANCY TEST IF WOMAN OF CHILD-BEARING AGE ***

## Management options[2,3]

**Conservative:** 80% stones pass on their own → analgesia, follow-up scan 2–3w, medical expulsive therapy, **safety-netting**

**ESWL (extra-corporeal shock wave lithotripsy):** US waves break up stone so it can pass spontaneously

**PCNL (percutaneous nephrolithotomy):** nephroscope into renal pelvis + lithotripsy to fragment stone

**Ureteroscopy + stone fragmentation:** endoscope up urethra & ureter, then stone lithotripsy & removal

---

[1] Alelign T, Petros B (2018) Kidney stone disease: an update on current concepts. *Adv Urol*, 2018:3068365.
[2] NICE (2019) *Renal and ureteric stones* [NG118]
[3] European Society of Urology (2021) *Guidelines on urolithiasis*

# Urinary tract malignancy

## Renal malignancy

**ADENOCARCINOMA / RENAL CELL CARCINOMA (85%)** = from cells of renal cortex

**Clinical presentation:** 50% = asymptomatic
- Haematuria = often **painless** (50%)
- Loin pain (40%) & **loin mass** (25%)
- Left-sided varicocele (less common)
- **Signs of complications:** anaemia, HTN, Cushing's, polycythaemia, pathological fractures (produces PTH-rp)

**Investigations:**
- **Hx & examination**
- **Urine dip:** MSU and cytology
- **Bloods:** U&Es, anaemia, ↑ ESR, ↑ ALP, ↑ PTH, ↑ Ca
- **Imaging:** USS & CT = solid mass

PROGNOSIS: 70% 5y survival (if mets <2y survival)

**Management:**
→ **Nephrectomy/nephron-ureterectomy**
→ ± Tyrosine kinase inhibitors ± immunotherapy (chemo/radio = less effective)

**Haematogenous spread to:**
Lung (cannonball mets), liver, bone, lymph nodes

**Risk factors for renal malignancy:**
1. **Prolonged haemodialysis**
2. Obesity, smoking, HTN, NSAIDs
3. Genetic: VHL, Birt–Hogg–Dubé

**TRANSITIONAL CELL CARCINOMA** (10%) = from cells of renal pelvis

**WILMS' TUMOUR** (20% of childhood malignancy) = undifferentiated mesoderm tumour

**Common presenting age:** <5y
**Symptoms:** flank pain, abdo mass, haematuria
**Management:** chemo + nephrectomy
**Prognosis:** 80% cure rate[4]

**NICE guidelines**

**Refer anyone ≥45y on a 2ww pathway if:**
- unexplained visible haematuria without UTI
- visible haematuria that persists after successful treatment of UTI

**Refer anyone ≥60y on a 2ww pathway if:**
- unexplained non-visible haematuria AND either dysuria OR a raised white cell count on a blood test

**Consider non-urgent referral for anyone ≥60 years with:**
- recurrent or persistent unexplained UTI

## Renal cysts

**CAUSES:**
→ Commonly develop with age (solitary or multiple)
→ Autosomal dominant PCKD = common cause

**SYMPTOMS:**
- **Often asymptomatic** = found incidentally
- Mass/pain if large
- Occasionally haematuria
- PCKD causes 20% of end-stage CKD

**MANAGEMENT:** → must rule out RCC as these can appear cystic
- **Simple cyst:** no intervention
- **Complicated cyst:** further investigation & surgical intervention

## Summary of causes of renal tract obstruction

| Luminal | Mural | Extramural | |
|---|---|---|---|
| • Calculi<br>• Clots<br>• Tumour<br>• Sloughed renal papillae (DM, NSAIDs) | • Ureteric stricture (surgery, catheter, TB) | • Tumour<br>• Pregnancy<br>• Bowel adhesions | • Retroperitoneal fibrosis<br>→ 1° autoimmune<br>→ 2° to some drugs (e.g. methotrexate) |

---

[4] NICE (2015, updated 2021) *Suspected cancer: recognition and referral* [NG12]

## Bladder cancer

→ *>90% = transitional cell carcinomas*

**Risk factors for bladder cancer:**

1. **Smoking**
2. Age
3. Dyes/chemicals
4. FHx
5. Obesity
6. Gender (male)
7. Long-term catheter
8. Bladder calculi

### CLINICAL PRESENTATION:

- **Painless haematuria**
- **LUTS:** voiding / obstructive symptoms
- Recurrent UTIs

### INVESTIGATIONS:

1. Hx & examination – note RFs
2. Urine dip & MCS + cytology = **haematuria**
3. Flexible cystoscopy & biopsy = **gold standard**
4. USS or CT urogram to visualise kidneys
5. CT CAP with contrast / MRI for staging if cancer found

### MANAGEMENT[5]:

**T1 (*in situ*): mucosa only**
→ transurethral resection of bladder tumour (TURBT)
+ Mitomycin C ± intravesical BCG course

**T2/3 (high grade): muscle invasion**
→ pre-op chemo + radical cystectomy or radiotherapy

**T4: invades past bladder** → chemotherapy

**Carcinoma *in situ*:** intravesical BCG course

## Prostate cancer

→ ***adenocarcinomas*** *most often located in the peripheral zone of the prostate*

**Very common:** approximately 1 in 9 men

**Risk factors for prostate cancer:**

1. **Age**
2. FHx
3. Black/Afro-Caribbean

**PSA:** SENSITIVE NOT SPECIFIC
= **also raised in BPH & UTI**

### CLINICAL PRESENTATION: often asymptomatic

- **Voiding symptoms** (hesitancy, poor stream, dribbling)
- ± **Haematuria** ± haematospermia
- **Red flags:** weight loss, night sweats, bone pain / pathological fractures (if metastases)

### INVESTIGATIONS:

1. **Hx & examination** → **DRE:** hard, craggy, irregular prostate
2. **Urinalysis** dip & MSU: (r/o infection)
3. **Bloods:** including PSA

**IF abnormal DRE &/or PSA raised for age:**
→ MRI + transrectal biopsy – Gleason grading
→ CT for staging
→ Bone scan if suspect mets

### MANAGEMENT[6]:

1. **Active surveillance:** if low risk disease
2. **Radical prostatectomy or radiotherapy:** if localised
3. **Surgical castration / androgen deprivation therapy:** if metastases
4. **Chemotherapy** if failed radical or hormonal treatment

---

[5] NICE (2015) *Bladder cancer* [NG2]
[6] NICE (2019, updated 2021) *Prostate cancer* [NG131]

# Bladder outlet obstruction

## Common causes

→ BPH
→ Prolapse
→ Post-incontinence surgery
→ Bladder calculi
→ Urethral strictures
→ Malignancy (bladder/urethral/prostatic)
→ External compression
→ Neurological disease

## Clinical presentation

rapid onset

1. **Lower urinary tract symptoms** (LUTS)
2. **Acute urinary retention:** suprapubic pain, palpable bladder, anuria
3. **Chronic urinary retention:** LUTS, renal impairment, palpable bladder, overflow incontinence, large residual urine volume

| Voiding symptoms | Storage symptoms |
|---|---|
| Hesitancy<br>Poor flow<br>Post-void dribbling<br>Dysuria<br>(sensation of incomplete emptying) | **F**requency<br>**U**rgency ± **u**rge incontinence 'FUN'<br>**N**octuria<br>(bedwetting / overflow Incontinence) |
| Present in **obstruction** | Present if **bladder dysfunction**<br>(can be 2° to chronic obstruction) |

## Bladder calculi

**PATHOGENESIS:** crystallisation of minerals in urine if it becomes concentrated (if incomplete emptying / urine stasis)

*Often uric acid or calcium stones (or struvite if UTI-related)*

**CAUSES:**

| Outlet obstruction | Neurogenic retention | Other |
|---|---|---|
| • BPH / prostate carcinoma<br>• Bladder tumour<br>• Urethral stricture<br>• Prolapse (in women) | • Stroke / spinal cord injury<br>• Spina bifida<br>• Diabetes<br>• Augmentation cystoplasty | • Infection (UTIs)<br>• Dehydration<br>• Foreign body / catheter<br>• Passage of renal calculi into bladder |

**CLINICAL PRESENTATION:**

- **LUTS:** voiding & storage
- **Dysuria & haematuria** at end of stream
- Lower abdo pain

± recurrent UTI

**INVESTIGATIONS:**

- **Hx & exam:** abdo & pelvic + DRE
- **Bloods:** FBC, CRP, U&Es, Ca, $PO_4$, urate, glucose, VBG ($HCO_3$)
- **Urine dip & MCS** – r/o infection
- **Basic imaging:** USS
- **Specialist imaging:** CT KUB

**MANAGEMENT[7]:**

**Transurethral cystolitholapaxy**

1. Cystoscope passed up urethra to identify stones
2. Crush stones or fragment with laser / pneumatic device
3. Bladder irrigation to remove fragments

**OR Open cystolithotomy** = surgical removal of bladder stones via a lower abdominal incision

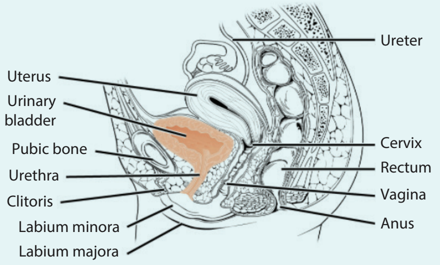


**Fig. 15.4** Female genitourinary anatomy.

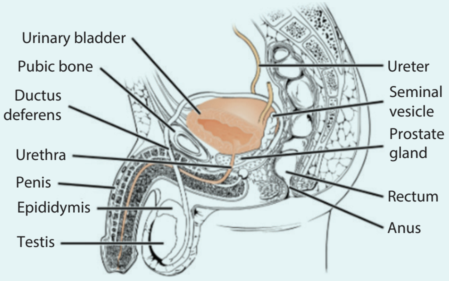


**Fig. 15.5** Male genitourinary anatomy.

**IPSS:** score/questionnaire to determine how much symptoms impact daily life

**Management of acute urinary retention:**

1. **ABCDE**
2. Bloods: FBC, U&Es (deranged U&Es = HPCR → need USS KUB & urology R/V)
3. **Catheter** (3-way if haematuria)
4. Full Hx and examination (including DRE & neuro exam)
5. Monitor fluid and electrolyte balance

Bladder calculi increase risk of bladder cancer

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[7] European Society of Urology (2021) *Guidelines on bladder stones*

## Benign prostatic hyperplasia (BPH)

*→ 50% of over 50s*
*→ affect transitional zone of prostate*

**PATHOGENESIS:** occurs **with age** due to **androgens** stimulating **increase in number of prostate cells**

**CLINICAL PRESENTATION:**

- **Voiding symptoms** ± secondary storage symptoms
- ± **Haematuria**
- **Enlarged, smooth prostate** on DRE
- **Acute retention** (sometimes occurs = rapid & painful)

**INVESTIGATIONS:**

1. **History & examination:** IPSS score & **remember DRE**
2. **Urinalysis:** dip & MCS
3. **Uroflowmetry:** <10ml/sec suggests obstruction
4. **Bloods:** including PSA
5. **Transrectal USS + biopsy:** definitive diagnosis

**MANAGEMENT[8]: depends on symptom severity**

| Mild | Moderate | Severe |
|---|---|---|
| 1. Reassurance<br>2. Lifestyle (fluid intake)<br>3. Follow-up | **Pharmacotherapy**<br>1. **Alpha blockers (e.g. tamsulosin)** = relax smooth muscle<br>→ **SEs:** retrograde ejaculation, postural hypotension, dizziness, headache<br>2. ± **5-alpha-reductase inhibitors (e.g. finasteride)**<br>= inhibits testosterone → DHT conversion<br>→ **pros:** ↓ prostate size<br>→ **cons:** take 6w to 6m to work<br>→ **SEs:** loss of libido, impotence<br>3. ± **Anti-cholinergics (e.g. oxybutynin/ solifenacin)**<br>In combination with above to treat 2° storage LUTS | **Surgery:**<br>1. **TURP** = gold standard<br>2. **Other:** e.g. laser/steam treatment<br>3. **Prostatic artery embolisation**<br>4. **Self-catheterisation / long-term catheter** |

**Chronic urinary retention**

Chronic obstruction can lead to **chronic urinary retention** → gradual stretching of bladder over years (painless)

- **Symptoms:** asymptomatic or LUTS, palpable bladder, overflow incontinence, large residual volume of urine
- **Complications:** UTIs, calculi, high pressure chronic retention (HPCR) → renal impairment & hydronephrosis

Possible complications of TURP:
**Acute:** bleeding, UTI

**Chronic:** retrograde ejaculation, impotence, incontinence, bladder neck stenosis / urethral stricture

---

[8] NICE (2010, updated 2015) *Lower urinary tract symptoms in men* [CG97]

# Bladder trauma

## Causes

**Accidental:** RTAs, falls, crush injury, blow to lower abdomen → often concurrent **pelvic #** & other intra-abdo injury
→ **RF:** full bladder

**Iatrogenic:** complex/emergency abdominal & pelvic surgeries
→ **RF:** previous surgery, inflammation, malignancy

## Clinical presentation

- Haematuria / dysuria / inability to void
- Suprapubic tenderness
- Abdo distension / ileus / scrotal / perineal swelling

## Diagnosis/investigations

1. **Bloods**
2. **Retrograde cystography or CT cystography** →bladder filled with dye & scanned

## Management

**General:** observation, bladder drainage, prophylactic ABX

1. **Intraperitoneal rupture:** surgical repair
2. **Extraperitoneal rupture:** catheter until cystography shows resolution

### Types of injury:

1. **Contusion:** bruising
2. **Intraperitoneal rupture:** e.g. tear at dome of bladder
   = urine into abdo cavity → **peritonitis**
3. **Extraperitoneal rupture:** e.g. posterior tear
   = urine into surrounding tissues

### Urethral trauma:

**Causes** = penile fractures, 'straddle injuries' e.g. bike

→ **Sx:** similar to bladder trauma
→ **Mx:** surgery if severe, catheterise & monitor if mild

**Posterior urethral trauma** = more common

# Scrotal lumps

**Summary of scrotal lump DDx**

| | |
|---|---|
| **Painless lump** | • Testicular tumour<br>• Hydrocele |
| **Painful lump** | • Testicular torsion<br>• Varicocele<br>• Epididymo-orchitis<br>• Epididymal cyst<br>• Torted hydatid of Morgagni<br>• Strangulated inguinal hernia |

## Anatomy of the scrotum

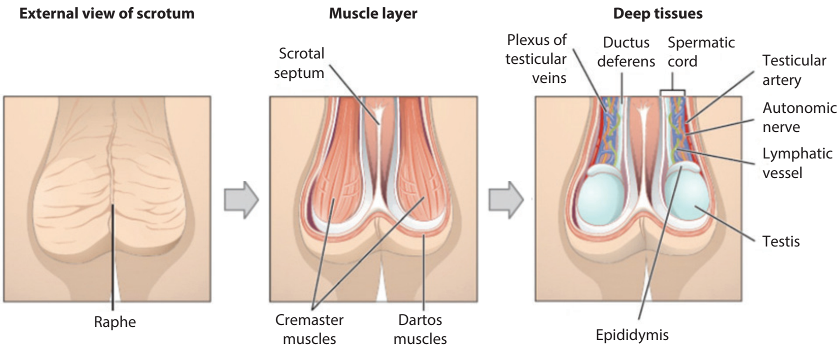
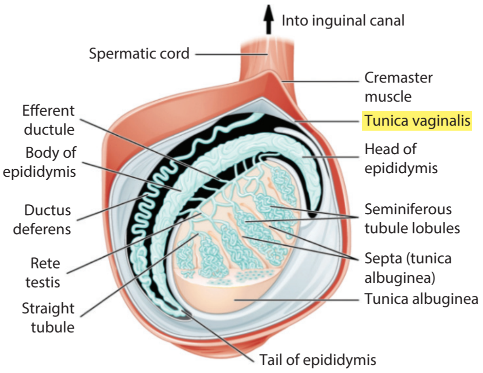

External view of scrotum
Raphe
Muscle layer
Scrotal septum
Cremaster muscles
Dartos muscles
Deep tissues
Plexus of testicular veins
Ductus deferens
Spermatic cord
Testicular artery
Autonomic nerve
Lymphatic vessel
Testis
Epididymis

**Fig. 15.6**


Into inguinal canal
Spermatic cord
Cremaster muscle
Tunica vaginalis
Efferent ductule
Body of epididymis
Head of epididymis
Ductus deferens
Seminiferous tubule lobules
Rete testis
Septa (tunica albuginea)
Straight tubule
Tunica albuginea
Tail of epididymis

**Tunica vaginalis:** part of peritoneum which descended with the testes

**Fig. 15.7**

## Differentials of a scrotal lump

| | Varicocele | Hydrocele | Epididymal cyst |
|---|---|---|---|
| **Pathogenesis** | Abnormal dilation of pampiniform plexus veins | Abnormal collection of peritoneal fluid between parietal & visceral layers of the tunica vaginalis | Benign fluid-filled sac arising from epididymis |
| **Causes** | 1. **Idiopathic valve incompetence**<br>Most = left side as left testicular vein is longer & enters renal vein at a right angle<br>2. **2° to left renal cell carcinoma:** must exclude if new varicocele | 1. **Communicating (patent processus vaginalis)**<br>→ congenital<br>→ ↑ IAP, fluid overload, dialysis<br>2. **Non-communicating (↑ fluid production or ↓ absorption)**<br>→ tumour or trauma<br>→ testicular torsion<br>→ epididymitis<br>→ idiopathic | 1. **Epididymal cyst:** collection of clear fluid<br>2. **Spermatocele:** collection of milky fluid that may contain sperm due to blocked outlet<br>e.g. post-vasectomy |
| **Presentation** | Dragging sensation / ache<br>**Complications:** subfertility | Usually painless | Tender<br>**Associations:** PCKD, CF, VHL |
| **Examination** | 'Bag of worms'<br>• Disappears when lie flat<br>• Positive Valsalva<br>→ **Dx:** with scrotal USS | • Fluctuant<br>• Anterior & inferior to testicle<br>• **Can get above mass**<br>• **Transilluminates** | • Superior & posterior to testicle<br>• Smooth & well-defined<br>• **Separate to testicle**<br>• **Transilluminates if very large** |
| **Management[9]** | **Embolisation / surgical ligation of veins if:**<br>1. Painful<br>2. Testicular atrophy or infertility<br>3. Child | **Congenital:** usually repaired if unresolved by 2y of age<br>**Adults:** surgical repair if symptoms<br>Needs Ix with USS to r/o tumour | 1. Reassurance<br>2. Ix with scrotal USS if unsure of diagnosis<br>3. Surgical excision if symptomatic |

---

[9] NICE CKS (2022) *Scrotal pain and swelling; Management scenarios*

## Epididymo-orchitis

→ *inflammation of epididymis ± testicle*

**CLINICAL PRESENTATION:** difficult to r/o torsion in many cases

- **Painful swelling** of epididymis ± testicle = sudden onset (h/d)
- Dysuria, sweats, fever
- **Hx of UTI/STI symptoms:** discharge/dysuria/LUTS

**ON EXAMINATION:**

- Unilateral scrotal swelling; tender epididymis ± testicle
- Erythematous
- **Phren's sign:** pain relief with elevation of hemi-scrotum

→ Can become tense & progress to form an abscess

**MANAGEMENT[10]:**

1. **Analgesia** ± scrotal support
2. **Antibiotics**
   - → **STI:** doxycycline ± ceftriaxone
   - → **UTI:** ciprofloxacin

**INVESTIGATIONS:**

1. Hx (sexual/LUTS)
2. Examination (abdo, penis, testicles, DRE)
3. **First catch urine:** MCS & STI screen
4. USS (shows ↑ blood to epididymis)

**Organisms:**

**STIs** (chlamydia, gonococcal)
**UTIs** (*E. coli*, pseudomonas)

**Rarer causes:** mumps, TB, amiodarone

**Top DDx = testicular torsion**

## Testicular torsion

**Pathogenesis:** *twisting of testes & spermatic cord → strangulation → ischaemia → necrosis = emergency*

**CLINICAL PRESENTATION:** neonates & those aged 10–30y

- **Sudden onset pain in testes** = unilateral
- ± Pain in loin or groin area
- Nausea/vomiting

**ON EXAMINATION:**

- **Extremely tender testes** = unilateral
- **May be red, hot, swollen**
- Testis is higher than normal & lie is transverse
- **Absent cremasteric reflex**

**MANAGEMENT[11]: urgent surgery** (ASAP – within 6h)

→ Scrotal exploration, detort testis & bilateral orchidopexy

→ If testis is non-viable then orchidectomy + orchidopexy of contralateral testis

**Causes of torsion:**

1. **Congenital abnormality:** bell clapper/undescended testes
2. Idiopathic

*High insertion of tunica vaginalis to spermatic cord enables testicular rotation*

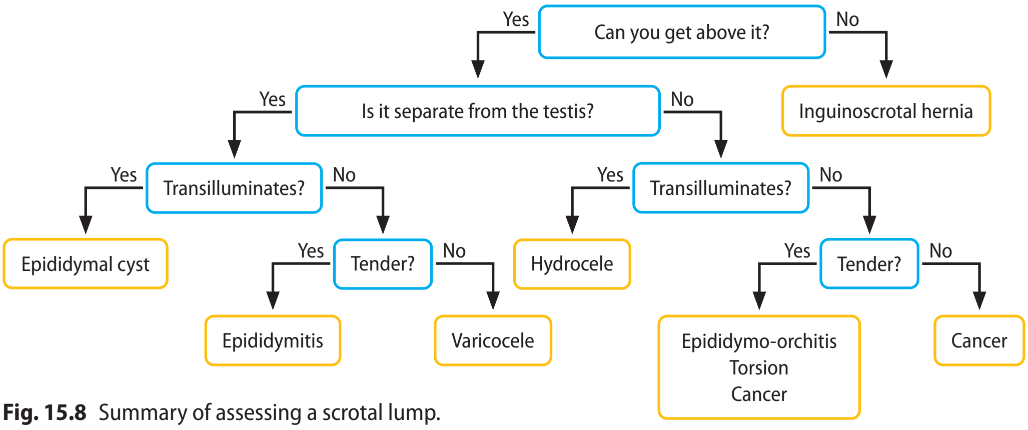

Can you get above it?
Yes
No
Inguinoscrotal hernia
Is it separate from the testis?
Yes
No
Transilluminates?
Yes
No
Epididymal cyst
Tender?
Yes
No
Epididymitis
Varicocele
Transilluminates?
Yes
No
Hydrocele
Tender?
Yes
No
Epididymo-orchitis
Torsion
Cancer
Cancer

**Fig. 15.8** Summary of assessing a scrotal lump.

---

[10] NICE CKS (2020) *Epididymo-orchitis; Management scenario*

[11] European Society of Urology (2016) *Guidelines on paediatric urology*

# Testicular tumours

## Classification of testicular tumours

**PRIMARY TUMOURS**

- **GERM CELL TUMOURS** (~90%)
  - **NON-SEMINOMA** (~50%)
    - → Mixed
    - → Choriocarcinoma
    - → Yolk sac tumour
    - → Embryonal carcinoma
    - → Teratoma
    - **Peak age:** late teens to 30y<br>**Spread:** haematological<br>**Marker:** hCG & AFP
  - **SEMINOMAS** (~50%) = from seminiferous tubules
    - **Peak age:** 30–50y<br>**Spread:** lymphatics<br>**Marker:** hCG & LDH
- **NON-GERM CELL TUMOURS**
  - → Leydig cell
  - → Sertoli cell
  - → Gonadoblastoma
  - **Secrete testosterone / oestrogen** causing sexual characteristics / precocious puberty / ambiguous genitalia at birth

**SECONDARY TUMOURS:** lymphoma (older men), leukaemia, metastatic

## Clinical presentation

- Testicular lump = solid, fixed, irregular
- ± Ache/heaviness/dragging
- ± Secondary hydrocele
- **Metastatic Sx:** weight loss, back pain, dyspnoea

## Investigations

- **Hx & examination**
- **Bloods:** FBC, CRP, U&Es, LFTs
- **Tumour markers:** AFP, LDH & hCG
- **USS:** confirms Dx
- **CT, CXR** to rule out metastases

## Management

- Orchidectomy ± chemo ± radiotherapy
- 95% cure rate

**Remember testicular tumours as DDx in younger men**

**Risk factors for testicular tumours:**

- FHx
- Undescended testes
- White ethnicity
- HIV

# Conditions of the penis

## Phimosis

→ ***tight foreskin*** *which is difficult to retract*

**CAUSES:**

1. **Physiological:** normal in early childhood (most resolve by 3y of age)
2. **Pathological:** infection (balanitis/balanoposthitis), inflammation & scarring (balanitis xerotica obliterans)

**CLINICAL PRESENTATION:**

- Pain on intercourse/erections
- Difficulty urinating – dribbling, weak stream
- Infection
- Recurrent UTIs

**MANAGEMENT:** good hygiene, steroid cream ± circumcision

## Paraphimosis

→ ***foreskin retracted behind glans*** *& too tight to be replaced → venous obstruction + oedema →* ***emergency***

**CLINICAL PRESENTATION:** swollen, painful retracted behind glans

**MANAGEMENT[12]:**

1. **Analgesia**
2. **Swelling reduction:** compression, ice
3. **Manual reduction**
4. **'Puncture' technique** to drain oedema (urologists only)
5. **Dorsal slit** (urologists only)

**Commonly occurs when foreskin not replaced after:**
→ catheterisation
→ erection / sexual activity

## Peyronie's disease

→ ***curvature of the penis*** *(due to scar tissue in tunica albuginea)*

**CAUSES:** repeated microtrauma

**PRESENTATION:** curvature ± pain with erection

**MANAGEMENT:** often improves without Tx
→ Oral pentoxifyline / verapamil injections
→ Surgery if stopping sexual intercourse

**Risk factors for Peyronie's:** DM, HTN, Dupuytren's contracture, plantar fasciitis

## Priapism

→ ***prolonged unwanted erection >4h*** *in the absence of sexual desire or stimulation*[13]

**TYPES:**

**High flow:** ↑ arterial flow = less painful + semi-rigid (often post trauma) → ice packs & compression ± embolisation

**Recurrent/stuttering:** painful + often self-limiting in sickle cell disease → treated similarly to sickle crisis

**Low flow / ischaemic:** ↓ venous drainage = **painful** + rigid → aspiration ± shunt surgery

**LOW FLOW MANAGEMENT[14]:**

1. Observation **(up to 4h)**
2. Intracavernosal sympathomimetic (e.g. phenylephrine) ± aspiration ± shunt

**Risk factors for priapism:** SCD, leukaemia, trauma, cocaine, cannabis, antidepressants, pelvic tumours

---

[12] BMJ Best Practice (2022) *Paraphimosis*
[13] Reynard J, *et al.* (2019) *Oxford Handbook of Urology* 4e
[14] BMJ Best Practice (2021) *Priapism*

## Carcinoma of the penis

**CLINICAL PRESENTATION:**
burning, itching, ulceration, bleeding

**MANAGEMENT:**
glansectomy or partial / total penectomy

most commonly SCC (95%)

**Risk factors for carcinoma of the penis:**
phimosis, HPV (16 & 18), age

## Urethral stricture

→ ***narrowing of urethra*** *due to subepithelial scar tissue*

**CAUSES:**

1. **Iatrogenic:** e.g. catheterisation, prostatectomy
2. **Previous trauma:** e.g. saddle injury / pelvic fracture
3. **Infection (urethritis):** e.g. gonococcal/chlamydia
4. **Inflammatory conditions:** lichen sclerosus

**PRESENTATION:**
**Obstructive:** hesitancy, poor stream, dysuria → may present as an emergency with **acute retention**

**DIAGNOSIS:** uroflowmetry / cystoscopy / urodynamic studies

**MANAGEMENT:** endoscopic dilation or urethroplasty

## Urethritis

→ ***inflammation of the urethra*** *due to infection*

**PRESENTATION:**

- **Dysuria** + frequency/urgency
- Discharge/itching
- Pain during sex

**INVESTIGATIONS:**

1. **Hx & examination**
2. **First catch urine:** dip & MSU
3. **Urethral swab:** polymorphonuclear leukocytes on microscopy

**MANAGEMENT:**
**Antibiotics** – depending on causative organism and local guidelines

**Common organisms in urethritis:**
*Neisseria gonorrhoeae* and *Chlamydia trachomatis**

*Chlamydia may present as *Reiter's triad* (urethritis + reactive arthritis + uveitis)

## Erectile dysfunction (impotence)

**CAUSES:**

Impotence = common

1. **Organic causes:**
→ **Smoking, alcohol, diabetes** ('big 3' organic causes)
→ **Other:** hypogonadism, hyper-/hypothyroidism, spinal cord lesion, MS, hypertension, prostate radiotherapy, drugs

2. **Psychogenic causes** (often still achieve morning erection):
→ Rule out organic causes first

**INVESTIGATIONS:** r/o organic cause

- Full sexual & psychological history
- **Bloods:** U&Es, LFT, glucose, lipids, TFT
- **Hormones:** testosterone, LH, FSH, prolactin
- ± **Doppler scan** ± **nocturnal penile tumescence testing**

**MANAGEMENT[15]:**

1. Treat underlying cause / **lifestyle factors**
2. Psychological support / counselling
3. **Phosphodiesterase-5 inhibitor** e.g. sildenafil (Viagra)
4. **Other options:** vacuum expanders, tumescence devices, intracavernosal injection, penile prosthesis

**Drugs causing impotence:**

- Beta-blockers
- Antipsychotics
- Diuretics
- Antidepressants

**Phosphodiesterase-5 inhibitors**
→ take up to 4h before sex
→ still requires arousal
→ food & alcohol reduce absorption
→ no more than 1 per day

**Contraindications for phosphodiesterase inhibitors**

- SBP <90
- Concurrent nitrates
- Recent stroke/angina
- Recent MI

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[15] NICE CKS (2020) *Management of erectile dysfunction; Management scenarios*

# 16

# CRITICAL ILLNESS

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**The semi-conscious/unconscious patient** .......... 207
**Organ support** .......... 208

## ABBREVIATIONS

**AED** – Anti-epileptic drug
**ARDS** – Acute respiratory distress syndrome
**AVM** – Arteriovenous malformation
**AVPU** – Alert, verbal, pain, unresponsive
**BG** – Blood glucose
**BiPAP** – Bi-level positive airway pressure
**BLS** – Basic life support
**BZD** – Benzodiazepine
**CBG** – Capillary blood glucose
**CO** – Cardiac output
**COC** – Ceiling of care
**CPAP** – Continuous positive airway pressure
**CPR** – Cardiopulmonary resuscitation
**CVC** – Central venous catheter
**DCC** – Direct current cardioversion
**DCML** – Dorsal column medial lemniscus
**DKA** – Diabetic ketoacidosis
**DNACPR** – Do not attempt CPR
**ECMO** – Extra-corporeal membrane oxygenation
**FiO$_2$** – Fraction of inspired oxygen
**GCS** – Glasgow Coma Score/Scale
**GI** – Gastrointestinal
**HDU** – High dependency unit
**HHS** – Hyperglycaemic hyperosmolar state
**ICP** – Intracranial pressure
**ICU** – Intensive care unit
**LP** – Lumbar puncture
**NEWS** – National Early Warning Score
**NIV** – Non-invasive ventilation
**NRM** – Non-rebreather mask
**SAH** – Subarachnoid haemorrhage
**SpO$_2$** – Oxygen saturation
**SVR** – Systemic vascular resistance
**SVT** – Supraventricular tachycardia
**T2RF** – Type 2 respiratory failure
**VF** – Ventricular fibrillation
**VT** – Ventricular tachycardia
**WOB** – Work of breathing

# The critically ill patient

**Normal physiological parameters**

| RR | $SpO_2$ | HR | SBP | Temp |
|---|---|---|---|---|
| 12–20 breaths per min | >94% | 60–100 beats per min | 90–120 mmHg | 36.0–37.2°C |

## Assessment of the seriously ill patient (ABCDE)

**Airway**

- Assess patency – obstruction by foreign bodies, vomit, blood, tongue (loss of tone)

**Breathing** → inspect, listen, measure ($RR, SpO_2$), chest exam (palpate, percuss, auscultate)

- Effort – RR, WOB, accessory muscles, added sounds (wheeze/stertor/stridor/crackles)
- Efficacy – talking, air entry, expansion, $SaO_2$ (>92%), **ABG**
- Effects – skin colour, conscious level

**Circulation** → look, feel (for pulse), measure (HR, BP, CRT)

- Heart – **pulse:** rate, rhythm, **auscultate:** murmurs
- Blood pressure – hypotension = **late sign**
- Capillary refill (<2s)
- Peripheral temp., colour
- Urine output

**Disability (D → G = Don't Ever Forget Glucose)**

- Level of consciousness (AVPU/GCS)
- Pupils – size, reactivity
- Posture & tone
- **Blood glucose**

**Exposure:** head to toe assessment

- Rash, injuries, bruises, abdo tenderness, calf tenderness
- Pain (GCS) – consider analgesia

## Management of the seriously ill patient

**Airway & breathing:**

- Open & maintain airway – head tilt, chin lift / jaw thrust
- 100% high flow $O_2$ – via NRM
- Anaesthetist involvement – if need intubation (ET tube)
- Consider **CXR**

**Circulation** → 16G/14G

- Obtain IV access – 2 wide-bore cannulas in antecubital fossa
- Take bloods – FBC, U&Es, LFT, coag, trop, X-match ± cultures
- Fluid bolus – 500ml crystalloid over 10min
- **ECG**
- Consider catheterising – guide fluid resuscitation
- Consider inotropes/vasopressors

Continuous monitoring & reassessment is VITAL in the Mx of acutely ill patients

## Recognising the critically ill patient

**EARLY WARNING SCORES (NEWS2)[1]:** (not if <16y or pregnant)
→ score out of 20 as a predictor of death/survival

| Score | Risk | Response |
|---|---|---|
| **0–4** | Low | Ward-based |
| **3 in any category** | Low–Med | Urgent ward-based |
| **5–6** | Medium | Urgent (<60min) |
| **≥7** | High | Emergency (<30min) |

| Physiological parameter | Score | | | | | | |
|---|---|---|---|---|---|---|---|
| | 3 | 2 | 1 | 0 | 1 | 2 | 3 |
| Respiration rate (per minute) | ≤8 | | 9–11 | 12–20 | | 21–24 | ≥25 |
| $SpO_2$ Scale 1 (%) | ≤91 | 92–93 | 94–95 | ≥96 | | | |
| $SpO_2$ Scale 2 (%) | ≤83 | 84–85 | 86–87 | 88–92<br>≥93 on air | 93–94 on oxygen | 95–96 on oxygen | ≥97 on oxygen |
| Air or oxygen | | Oxygen | | Air | | | |
| Systolic blood pressure (mmHg) | ≤90 | 91–100 | 101–110 | 111–219 | | | ≥220 |
| Pulse (per minute) | ≤40 | | 41–50 | 51–90 | 31–110 | 111–130 | ≥131 |
| Consciousness | | | | Alert | | | CVPU |
| Temperature (°C) | ≤35.0 | | 35.1–36.0 | 36.1–38.0 | 38.1–39.0 | ≥39.1 | |

**Fig. 16.1** NEWS2 score.

**A score ≥7 warrants critical care team assessment for transfer to HDU**

**Scale 2:** if confirmed previous or chronic hypoxic resp. failure

What is baseline BP?
Do they take antihypertensives?

| | |
|---|---|
| **A** | Awake |
| **C** | Confusion |
| **V** | Verbal – responds to voice |
| **P** | Pain – responds |
| **U** | Unresponsive |

**Other useful parameters:**

1. **Capillary refill time** (<2s)
2. **Lactate** (0.5–1.5mmol/L)
3. **Urine output** (0.5ml/kg/h)

---

[1] RCOP (2017) *National Early Warning Score (NEWS) 2: Standardising the assessment of acute-illness severity in the NHS*

# The semi-conscious/unconscious patient

**An unconscious patient:** defined as a GCS of <8

**Coma:** a state of impaired consciousness, where a person is **unresponsive & cannot be woken**

## Approach to an unconscious patient

**Airway** – can they talk? any obstructions? insert airway if needed
**Breathing** – RR, auscultate, sats, ABG
**Circulation** – HR, BP, auscultate → **IV access & bloods**
**Disability** – pupils, AVPU, GCS, drug chart, **glucose**
**Exposure** & **escalate** – temp., rash

**KEY BLOODS:**

- **General:** FBC, CRP, U&Es, LFTs, TFTs, **glucose**
- **Sepsis:** blood cultures
- **Haemorrhage/trauma:** coagulation & X-match
- **Overdose:** toxicology screen

**OTHER INVESTIGATIONS:**

- **Imaging:** CT/MRI → if trauma / suspect bleed or mass lesion
- **LP:** if suspect meningoencephalitis/infection

**Pupil assessment:**

1. **Document size:** should be equal **(normally 2–4cm in light)**
2. **Document reaction:**

+ reacts – no reaction SL = sluggish reaction

**Pinpoint pupils:** opioid toxicity or pontine lesion
**Dilated pupils:** intracranial pathology (herniation, PCA aneurysm)
**Small & reactive pupils:** metabolic disturbance

## Glasgow Coma Score/Scale[2]

| Eye response | | Verbal response | | Motor response | |
|---|---|---|---|---|---|
| 4 | Open spontaneously | 5 | Orientated | 6 | Obeys commands |
| 3 | Open to speech | 4 | Confused | 5 | Localises to pain |
| 2 | Open to pain | 3 | Inappropriate words | 4 | Withdraws from pain |
| 1 | Do not open | 2 | Incomprehensible sounds | 3 | Flexes to pain (decorticate) |
| | | 1 | None | 2 | Extends to pain (decerebrate) |
| | | | | 1 | No response |

**Pain / noxious stimuli:**

- trapezius squeeze
- supraorbital pressure

**Differentials**

**Vascular:** intracerebral haemorrhage, SAH, stroke
→ preceding headache / focal neuro signs

**Metabolic:** hypo-/hyperglycaemia, hyponatraemia
→ check BG, Hx of DM

**Poisoning:** opiates, TCAs, BZDs, CO, alcohol
→ Hx of drug/alcohol use, mental health, pupils

**Infection:** meningitis, encephalitis, sepsis
→ fever, rash, prodrome, septic shock

**Head injury, status epilepticus, hypothermia**

**AVPU**

| | | |
|---|---|---|
| **A** | **Alert** | Responds spontaneously |
| **V** | **Verbal** | Responds to voice |
| **P** | **Pain** | Responds to pain |
| **U** | **Unresponsive** | No response |

**Interpreting GCS**

**Minimum score:** 3
**Maximum score:** 15

**13–15:** mild
**9–12:** moderate
**≤8:** severe

**'GCS ≤8 intubate'**

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[2] Glasgow Coma Scale – Glasgow University (GCS), and Sir Graham Teasdale (www.glasgowcomascale.org)

# Organ support

**The role of ICU:** ICU does not have alternative treatments/medications to wards, **but** can offer **organ support** for patients who it is thought will benefit

## Respiratory support

### 1. OXYGEN DELIVERY:

**Indications:** Type 1 respiratory failure (pneumonia, asthma, pulmonary oedema, PE, pneumothorax etc.)

| Delivery device | Flow & $FiO_2$ | Oxygen delivered |
|---|---|---|
| **Nasal cannulae** | **Flow rate:** 1–6L/min<br>**$FiO_2$:** 24–44% | 'Uncontrolled $O_2$ delivery' |
| **Hudson mask**<br>Rarely used (NRM instead) | **Flow rate:** 5–8L/min<br>**$FiO_2$:** 35–50% | 'Uncontrolled $O_2$ delivery' |
| **Non-rebreather mask**<br>Most commonly used | **Flow rate:** 8–15L/min<br>**$FiO_2$:** up to 80% | 'Uncontrolled $O_2$ delivery' |
| **Venturi mask**<br>Used if concern of hypoxic drive (COPD) or $CO_2$ retention | **Flow rate:** fixed, depends on valve<br>**$FiO_2$:** 24, 28, 31, 35, 40, 60%<br>(lower flow for lower $FiO_2$) | **Blue:** 24% = 2–4L/min<br>**White:** 28% = 4–6L/min<br>**Orange:** 31% = 6–8L/min<br>**Yellow:** 35% = 8–10L/min<br>**Red:** 40% = 10–12L/min<br>**Green:** 60% = 12–15L/min |
| **Nebuliser mask** | **Flow rate:** 6–8L/min<br>Delivery of nebulised drugs | |

### 2. CPAP (continuous positive airway pressure)

### 3. NON-INVASIVE VENTILATION (NIV) / BiPAP (bi-level positive airway pressure)

**Indications:** Type 2 respiratory failure (COPD exacerbation + acidosis, neuromuscular disease)

**Mechanism:** oxygen delivered at two alternating pressures to match inspiration & expiration

**Contraindications for CPAP/NIV:**

- facial trauma
- closed pneumothorax
- vomiting
- GCS <8

### 4. INTUBATION (invasive ventilation)

**Indications:** coma, refractory COPD exacerbation, ARDS, heart failure, pneumonia, sepsis, trauma

**Mechanism:** sedation & endotracheal tube insertion → only on ICU

### 5. ECMO (extra-corporeal membrane oxygenation) → highly specialised, only offered at a limited number of centres

**Indications:** severe illness/shock, causing insufficient respiratory or cardiac functioning

**Mechanism:** blood is pumped out of the body via 'artificial lungs', for $CO_2$ & $O_2$ exchange, then pumped back around the body

## Cardiac support

### 1. INOTROPES/VASOPRESSORS

**Indications:** low CO (e.g. cardiogenic shock, acute decompensated heart failure)

| Inotropes | Vasopressors | Both |
|---|---|---|
| Dobutamine | Metaraminol + phenylephrine | Adrenaline |
| Milrinone | Vasopressin | Dopamine |
| | Noradrenaline | |

Increasing effect on CO/SVR

**Mechanism:**

- **Inotropes:** increase cardiac output (CO)
- **Vasopressors:** increase systemic vascular resistance (SVR)

**2. MECHANICAL SUPPORT** = IABP (intra-aortic balloon pump), VAD (ventricular assist device)

**3. ECMO:** veno–arterial supports heart & lungs, veno–veno supports just lungs

## Renal support

### RENAL REPLACEMENT THERAPY (RRT)

**Indications:** refractory acidosis, hyperkalaemia, uraemia, fluid overload

**Mechanism:** continuous veno–venous haemodiafiltration or intermittent haemodialysis which augment electrolyte & fluid homeostasis

## Neurological support

### SEDATION & NEUROPROTECTIVE MEASURES

- Maintain optimal oxygenation, ventilation, blood pressure, blood sugar, body temperature & electrolyte levels
- Intracranial pressure (ICP) monitoring
- Seizure prophylaxis & treatment

## Other

- Thermoregulation
- Plasma exchange
- Analgesia
- End-of-life care
- Organ donation

**Ceiling of care (COC):** how aggressively a patient will be treated

**Full escalation:** no limitations placed on active treatment – would be for discussion with ICU
**Ward-based:** everything on a conventional ward but **not for ICU**
**Palliative:** everything to ensure comfort, **no active treatment**

COC decisions are made based on the clinical picture and reversibility, a patient's baseline function, and any advance directives / DNACPR documentation

**Cardiac monitoring**

- arterial lines
- continuous cardiac monitor
- echo

# EMERGENCY PRESENTATIONS

# 17

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## ABBREVIATIONS

**ABG** – Arterial blood gas
**ACEi** – Angiotensin-converting enzyme inhibitor
**AED** – Anti-epileptic drug
**AF** – Atrial fibrillation
**ARB** – Angiotensin receptor blocker
**AVPU** – Alert, verbal, pain, unresponsive
**BB** – Beta-blocker
**BBB** – Bundle branch block
**BE** – Base excess
**BG** – Blood glucose
**BLS** – Basic life support
**BNP** – B-type natriuretic peptide
**BZD** – Benzodiazepine
**CBG** – Capillary blood glucose
**CBK** – Capillary blood ketones
**CCB** – Calcium channel blocker
**CK** – Creatine kinase
**CKD** – Chronic kidney disease
**COPD** – Chronic obstructive pulmonary disease
**CPR** – Cardiopulmonary resuscitation
**CRT** – Capillary refill time
**CVC** – Central venous catheter
**D&V** – Diarrhoea & vomiting
**DCC** – Direct current cardioversion
**DKA** – Diabetic ketoacidosis
**DM** – Diabetes mellitus
**G&S** – Group & save
**GCS** – Glasgow Coma Score/Scale
**GI** – Gastrointestinal
**GTN** – Glyceryl trinitrate
**HDU** – High dependency unit
**HF** – Heart failure
**HHS** – Hyperglycaemic hyperosmolar state
**ICU** – Intensive care unit
**IVDU** – Intravenous drug user
**JVP** – Jugular venous pressure
**KCl** – Potassium chloride
**LMWH** – Low molecular weight heparin
**MI** – Myocardial infarction
**NAC** – *N*-acetylcysteine
**NIV** – Non-invasive ventilation
**NRM** – Non-rebreather mask
**PCI** – Percutaneous coronary intervention
**PE** – Pulmonary embolism
**PEA** – Pulseless electrical activity
**PEF** – Peak expiratory flow
**PVT** – Pulseless ventricular tachycardia
**RTA** – Renal tubular acidosis
**SABA** – Short-acting beta agonist
**SAMA** – Short-acting muscarinic antagonist
**SOB** – Shortness of breath
**STEMI** – ST elevation myocardial infarction
**SVT** – Supraventricular tachycardia
**TCA** – Tricyclic antidepressant
**UO** – Urine output
**UTI** – Urinary tract infection
**VBG** – Venous blood gas
**VF** – Ventricular fibrillation
**VT** – Ventricular tachycardia

# Acid–base disorders

**$FiO_2$:** fraction of inspired $O_2$ (21% or 0.21 in air)

**$PaO_2$:** partial pressure $O_2$ in arterial blood
**Depends on:**
- $FiO_2$
- ventilation & ventilation-perfusion matching

**$SaO_2$:** proportion of oxygenated Hb in arterial blood
**Depends on:** $O_2$–Hb dissociation curve

**$CaO_2$:** content of $O_2$ in arterial blood
**Depends on:** $SaO_2$ & Hb conc.

**Base excess (BE):** how much acid is needed to normalise pH
**Base deficit:** how much base is needed to normalise pH

→ Acid–base balance is maintained by the kidneys & pulmonary system
→ It can be assessed via venous or arterial blood gas

## Interpreting blood gases

### 1. Determine acid–base status

- **Acidaemia:** pH <7.35 → due to ↑ $PaCO_2$ or ↓ $HCO_3^-$
- **Alkalaemia:** pH >7.45 → due to ↓ $PaCO_2$ or ↑ $HCO_3^-$

### 2. Assess oxygenation (if arterial):

- **Hypoxia:** inadequate tissue oxygenation
- **Absolute hypoxaemia:** $PaO_2$ <10kPa (<8kPa = severe)
- **Relative hypoxaemia:** $PaO_2$ less than expected for $FiO_2$
  - i.e. if on high flow $O_2$, would expect $PaO_2$ to be higher than in just room air
  - → **Calculate using:** $PaO_2$: $FiO_2$ (P:F ratio)
  - → **$PaO_2$ should roughly** = $FiO_2$% – 10kPa

### 3. Determine if respiratory or metabolic problem:

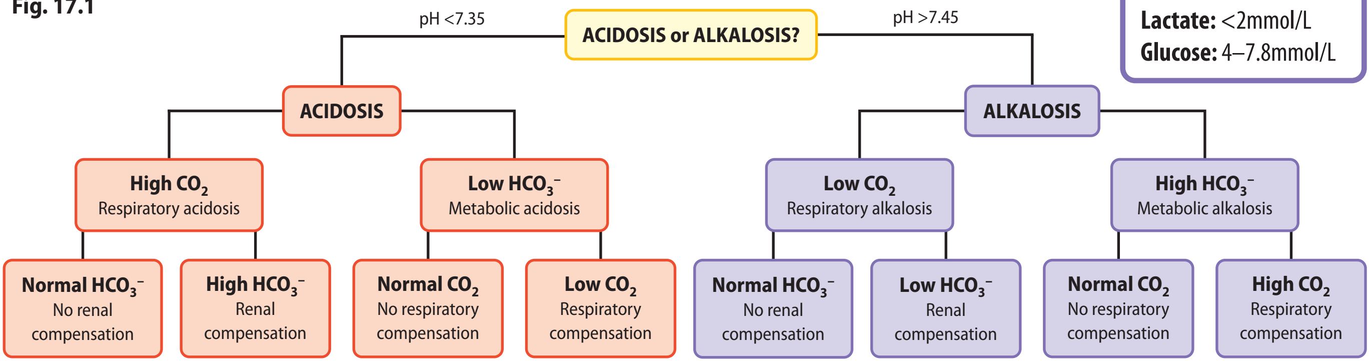
→ Is it the $PaCO_2$ or $HCO_3^-$ level that fits with the pH?

### 4. Determine if there is compensation:

→ Has the $PaCO_2$ or $HCO_3^-$ changed to oppose the pH?

**Normal values:**
**pH:** 7.35–7.45
**$PaCO_2$:** 4.5–6.0kPa
**$PaO_2$:** 9.5–14kPa
**$HCO_3^-$:** 22–26mmol/L
**BE:** –2 to +2
**Lactate:** <2mmol/L
**Glucose:** 4–7.8mmol/L

**Fig. 17.1**


ACIDOSIS or ALKALOSIS?
pH <7.35
pH >7.45
ACIDOSIS
ALKALOSIS
High $CO_2$ Respiratory acidosis
Low $HCO_3^-$ Metabolic acidosis
Low $CO_2$ Respiratory alkalosis
High $HCO_3^-$ Metabolic alkalosis
Normal $HCO_3^-$ No renal compensation
High $HCO_3^-$ Renal compensation
Normal $CO_2$ No respiratory compensation
Low $CO_2$ Respiratory compensation
Normal $HCO_3^-$ No renal compensation
Low $HCO_3^-$ Renal compensation
Normal $CO_2$ No respiratory compensation
High $CO_2$ Respiratory compensation

**Anion gap: $(Na^+ + K^+) - (Cl^- + HCO_3^-)$**
→ can help determine cause of acid–base imbalance

**Raised gap:** suggests extra $H^+$ from somewhere
e.g. lactate, ketones, uraemia, toxins

**Normal gap:** suggests $HCO_3^-$ loss
*e.g. diarrhoea, vomiting, RTA, Addison's*

**Normal:** 7–16mEq/L

### 5. Assess lactate: produced in anaerobic respiration

→ can indicate poor tissue perfusion
→ can also be caused by some drugs (salbutamol or metformin)

### 6. Assess blood glucose:

- may be cause of ABG problem (e.g. DKA)
- may be result of ABG problem (e.g. sepsis, liver disease)
- may impact on outcome (e.g. brain injury)

## Examples of acid–base disorders

| **Primary respiratory acidosis** (hypoventilation) | **Metabolic acidosis** | **Respiratory alkalosis** (hyperventilation) | **Metabolic alkalosis** |
|---|---|---|---|
| **Uncompensated:** Asthma, opioids, BZD | **Uncompensated:** Sepsis | **Uncompensated:** Pain, anxiety, PE, pneumonia, pneumothorax | **Uncompensated:** GI losses (D&V) |
| **Compensated:** COPD | **Compensated:** DKA, renal disease, sepsis | **Compensated:** Neuro disease | |

# Electrolyte abnormalities

## **Hyperglycaemia** (DKA & HHS)

### DIAGNOSIS:

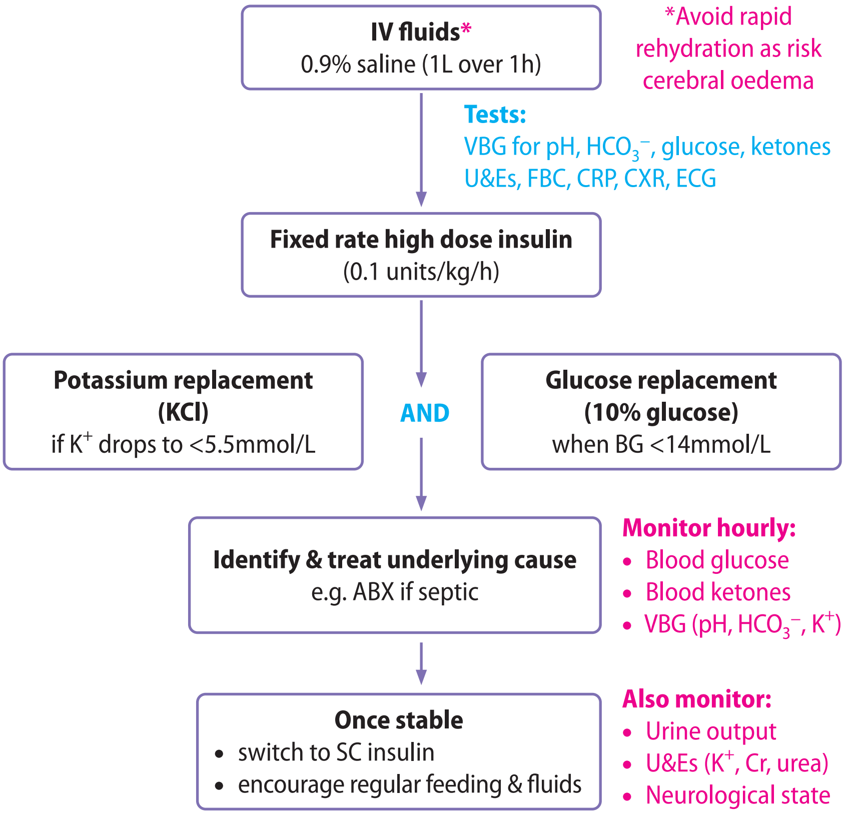
| DKA | HHS |
|---|---|
| 1. **Hyperglycaemia:** random BG >11mmol/L<br>2. **Ketosis:** capillary ketones >3mmol/L or urinary ketones ++<br>3. **Acidosis:** HCO$_3^-$ <15 or pH <7.35 | 1. **Profound hyperglycaemia:** random BG >30mmol/L<br>2. **No ketosis & pH >7.3**<br>3. **High osmolality:** >320mosmol/kg |

### MANAGEMENT OF HYPERGLYCAEMIC HYPEROSMOLAR STATE (HHS)[1]

1. **ABCDE** & confirm Dx with lab results & osmolality (VBG)
2. **IV FLUIDS** 0.9% saline (infusion over 24h)
3. **Do not start insulin** (until rate of fall in glucose is <5mmol/L per hour)
   → then give fixed rate insulin at **half dose of** DKA (0.05 units/kg/h)
4. **K$^+$ replacement with KCl** if K$^+$ drops to <5.5mmol/L
5. **Prophylactic anticoagulation** – LMWH for full duration of admission

### MANAGEMENT OF DIABETIC KETOACIDOSIS (DKA)[2]

1. **ABCDE**
2. **Confirm Dx with lab results** (VBG)
3. **Consider HDU if:**
   - CBK >6mmol/L
   - HCO$_3^-$ <5mmol/L
   - pH <7.0
   - GCS <12


**IV fluids***
0.9% saline (1L over 1h)
**Fixed rate high dose insulin**
(0.1 units/kg/h)
**Potassium replacement (KCl)**
if K$^+$ drops to <5.5mmol/L
**AND**
**Glucose replacement (10% glucose)**
when BG <14mmol/L
**Identify & treat underlying cause**
e.g. ABX if septic
**Once stable**
• switch to SC insulin
• encourage regular feeding & fluids

*Avoid rapid rehydration as risk cerebral oedema

**Tests:**
VBG for pH, HCO$_3^-$, glucose, ketones
U&Es, FBC, CRP, CXR, ECG

**Monitor hourly:**
- Blood glucose
- Blood ketones
- VBG (pH, HCO$_3^-$, K$^+$)

**Also monitor:**
- Urine output
- U&Es (K$^+$, Cr, urea)
- Neurological state

**Fig. 17.2** DKA management in adults.

---

[1] Diabetes UK (2012) *The management of hyperosmolar hyperglycaemic state (HHS) in adults with diabetes*

[2] Diabetes UK (2021) *The management of diabetic ketoacidosis in adults*

## Hypoglycaemia[3]

**Hypoglycaemia** = blood glucose <4mmol/L

### CAUSES:

- Reduced oral intake
- Intense exercise
- Binge drinking
- Taking too much insulin (if diabetic)

### SYMPTOMS:

- Irritable, anxious, confused
- Hungry or nauseous
- Shaky, dizzy, light-headed
- Seizures if severe

### MANAGEMENT:

1. Oral rapid-acting glucose **or** IV glucose 100ml 20% / IM glucagon 1mg
2. Long-acting carbohydrate e.g. toast (once CBG >4mmol/L)
3. **Look for cause** (document, monitor and get specialist review if needed)

## Hyperkalaemia

**Hyperkalaemia** = $K^+$ >5.2mmol/L
**Severe hyperkalaemia** = $K^+$ >6mmol/L

### CAUSES:

- CKD / renal failure
- Drugs (ACEis/ARBs, NSAIDs, spironolactone, potassium supplements)
- Burns / trauma / tissue injuries
- Hormonal disorders e.g. Addison's

### SYMPTOMS:

- Chest pain / palpitations
- Dizziness/weakness
- Abdominal pain / vomiting

### INVESTIGATIONS:

- U&Es
- ECG – tented T waves, flattened P wave, shortened QT interval

### MANAGEMENT:

1. 10ml 10% calcium gluconate
2. IV insulin + 25g glucose
3. Salbutamol nebs
4. Treat cause

---

[3] NICE BNF – *Treatment summary: Hypoglycaemia*

# Overdoses

## What to establish from the history

- What taken, how much and when?
- Route of exposure
- Whether exposure is a single overdose, staggered or chronic
- Any other agent(s) involved, including co-agents (e.g. alcohol, paracetamol)

## Investigations

1. ABCDE – check for other wounds/ injuries, check pupils & GCS
2. ECG
3. Urine sample – **drugs screen**
4. Bloods – FBC, U&Es, LFTs, **ABG** + specific toxin levels (e.g. salicylate)

## Management

### GENERAL MANAGEMENT:

1. Stabilise with ABCDE
2. Consider activated charcoal (reduced absorption **if <1h**)
3. Consider antidote if available
4. Increase elimination – multiple doses of activated charcoal **or** urine alkalinisation (salicylates) **or** dialysis
5. Monitoring & psychiatry assessment
6. Consider discussion with Poisons Centre in complex cases

**Check TOXBASE[4] for management of specific toxins**

| Poison | Effects | Management |
|---|---|---|
| **Alcohol** | • Hypoglycaemia<br>• Coma<br>• Respiratory failure | • Check blood alcohol levels<br>• Monitor blood glucose (IV glucose if need)<br>• Ventilatory support |
| **Acids/alkalis** | • Inflammation & ulceration of GI tract | • Early endoscopy |
| **Ethylene glycol** (antifreeze) | • Tachycardia<br>• Metabolic acidosis<br>• Renal failure | • **Fomepizole**<br>• Haemodialysis |
| **Paracetamol** | **24–48h:** abdo pain, vomiting<br>**3–5d:** liver failure (deranged LFTs, coagulopathy) | • Measure plasma paracetamol levels after 4h<br>• Activated charcoal if <1h<br>• **IV *N*-acetylcysteine** (if conc. >150mg/kg)* |
| **Aspirin (salicylates)** | **Within 4h:**<br>• N&V, drowsiness, blurred vision, tinnitus<br>• Hyperventilation (respiratory alkalosis → metabolic acidosis)<br>• Acute renal failure | • Measure plasma salicylate conc. (every 2h)<br>• Activated charcoal if <1h<br>• Urine alkalinisation<br>• Rapid BG, blood gases, Cr, FBC, ECG<br>• Supportive Tx: fluids, dialysis etc.<br>• **No antidote** |
| **Iron** | **Initial:** D&V, haematemesis, melaena, gastric ulcers<br>**Latent period**<br>**6h later:** drowsy, coma, shock, convulse, liver fails | • Measure serum iron levels<br>• Gastric lavage if <1h not charcoal<br>• **IV deferoxamine** (chelates iron) |
| **Methadone / opioids** | • Drowsiness, pinpoint pupils<br>• Hypotension, bradycardia<br>• Resp depression (respiratory acidosis) | • Activated charcoal even > 1h<br>• IV fluids<br>• **IV/IM naloxone** (bolus then infusion) |
| **TCAs** | • Tachycardia, arrhythmias, hypokalaemia, wide QRS<br>• Dry mouth, blurred vision, urinary retention<br>• Agitation, confusion, convulsions, dilated pupils<br>• Respiratory depression | • Correct metabolic acidosis & hypokalaemia<br>• Ventilatory support<br>• Activated charcoal even if >1h<br>• **IV sodium bicarbonate** if arrhythmias<br>• Diazepam if convulsions |
| **BZDs** | • Sedation/drowsiness/coma<br>• Respiratory depression<br>• Hypotension, hyporeflexia | • Ventilatory support<br>• Consider **IV flumazenil** |

*use paracetamol nomogram to determine if NAC is required. NAC is started in all staggered overdoses.

---

[4] www.toxbase.org

# Seizures[5]

## Initial management

**ABCDE**

1. Secure airway if needed due to low GCS & give $O_2$
2. IV access to take bloods (FBC, U&Es, Ca, LFT, eGFR, ABG, AED levels)
3. **Take a CBG:** **D**on't **E**ver **F**orget **G**lucose (r/o hypoglycaemia)
4. Confirm it is an epileptic seizure (Hx, features, blood results etc.)

**In all cases of seizure, must consider a serious cause**

- Infection / viral encephalitis
- Tumour
- Large stroke
- Venous sinus thrombosis
- Toxins (drugs/alcohol)
- Metabolic (hypoglycaemia/calcaemia/magnesaemia)
- Autoimmune diseases

## Investigations

- Brain scans
- Lumbar puncture
- Bloods / septic screen
- AED levels
- Toxicology screen

## Manage convulsion

1. **IV lorazepam 4mg** → repeat in 5–10min (10mg buccal midazolam / rectal diazepam = 1st line in community)
2. **Alert a senior** for support
3. **IV AED (e.g. sodium valproate / phenytoin / levetiracetam)** → check Trust guidelines
4. **Alert critical care services (ICU/anaesthetics) for airway support & escalation of care**
5. Intubation & sedation

**IV BZD → IV AEDs → Anaesthetic + ICU**

**Status epilepticus[6]:**

- A seizure lasting ≥5min **OR**
- >1 shorter seizure without resolution between **OR**
- Repeated seizures for ≥30min

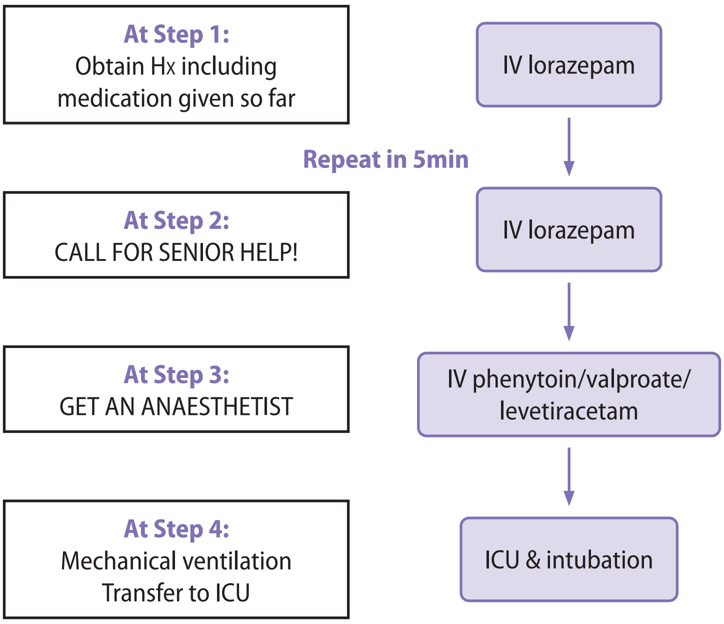

At Step 1:
Obtain Hx including medication given so far
IV lorazepam
Repeat in 5min
At Step 2:
CALL FOR SENIOR HELP!
IV lorazepam
At Step 3:
GET AN ANAESTHETIST
IV phenytoin/valproate/levetiracetam
At Step 4:
Mechanical ventilation
Transfer to ICU
ICU & intubation

**Fig. 17.3**

Ensure all AEDs are prescribed correctly

## Post-seizure care

1. **Start maintenance AED:** valproate or levetiracetam
2. **Continue patient's current AEDs:** if no oral route, give IV or via NG tube
3. **Refer to neurology**

---

[5] Association of Anaesthetists (2022) *Management of status epilepticus*

[6] Epilepsy Foundation definition of status epilepticus (www.epilepsy.com)

# Sepsis

→ life-threatening organ dysfunction due to dysregulated host response to infection

**Septic shock:** subset of sepsis where particularly profound circulatory, cellular & metabolic abnormalities substantially increase mortality

- **Signs:** sepsis **and** hypotensive (<90mmHg) despite fluid resus **and** lactate >2mmol/L
- **Mx:** SEPSIS 6 + vasopressors in ICU

**20–24% mortality**

**Neutropenic sepsis:** time-critical sepsis occurring in **immunocompromised patients** (e.g. chemotherapy)

- **Signs:** neutrophils <0.5 × $10^9$/L ± usual signs (often occult)
- **Mx:** ABCDE & SEPSIS 6 ASAP **(if suspect, don't wait for blood results)**

## When to trigger sepsis screen / assessment for red flags?

**NEWS2 ≥5 combined with ≥1 of:**

- Age >75y
- Impaired immunity (chemo, DM)
- Recent invasive procedure (surgery, trauma)
- Broken skin (indwelling lines, IVDU, cuts/burns)

**Or otherwise unexplained concern from staff/carer:**

- looks very unwell
- unexpected deterioration
- not responding to interventions

**Common sources of sepsis & associated signs/symptoms:**

- Pneumonia – consolidation, SOB, productive cough
- UTI – dysuria, urine dip
- Abdomen – tenderness
- Skin / soft tissue, bone/joint – warmth, swelling

## Risk stratification of sepsis

1. **SOFA/qSOFA:** official diagnostic criteria (increase of ≥2 from baseline)
2. **Red flag criteria:** not 'formal diagnosis' but suggests high likelihood of organ dysfunction (**pragmatic approach**)

Risk stratificaton tools help determine initial management but do not replace clinical judgement

**qSOFA score**
**H**ypotension SBP <100
**A**ltered mental state
**T**achypnoeic >22

| Red flags (any = start SEPSIS 6) | Amber flags (any = need further review/Ix) |
|---|---|
| Objective evidence of new/altered mental state | Relatives concerned over mental state/ functioning |
| SBP ≤90mmHg | SBP 91–100mmHg |
| HR ≥130bpm | HR 91–130bpm |
| RR ≥25bpm | RR 21–24bpm |
| Needs $O_2$ for $PaO_2$ ≥92% (≥88% COPD) | Temperature <36°C |
| Non-blanching rash / mottled skin | Clinical signs of wound infection |
| Lactate ≥2mmol/L | Immunosuppressed |
| Oliguria <0.5ml/h or anuria for 18h | Trauma/surgery in past 8w |

## Managing sepsis[7]

SEPSIS 6 within 1h

1. **Ensure senior clinician attends**
2. **Oxygen** if sats <92%; aim for 94–98% (88–92% in known $CO_2$ retention)
3. **IV access:** take bloods: culture ×2, glucose, lactate, FBC, U&Es, CRP, clotting; consider sampling other sites
4. **IV antibiotics:** appropriate dose broad-spectrum therapy
5. **IV fluids:** bolus of 500ml; repeat if clinically indicated up to maximum 20ml/kg
6. **Monitor:** use NEWS2; urine output (catheterise) & fluid balance chart; repeat ABG/VBG regularly if abnormal

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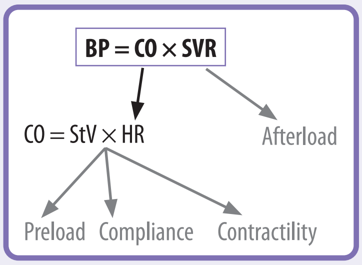
[7] NICE (2016, updated 2017) *Sepsis* [NG51]

# Shock

→ Life-threatening failure of oxygen delivery to tissues



**CO** = cardiac output; **HR** = heart rate; **StV** = stroke volume; **SVR** = systemic vascular resistance

## General signs of shock

1. **Low BP**
2. **Signs of ↓ perfusion**
   - ↑ cap refill
   - cool peripheries
   - ↑ RR
   - ↓ urine output
   - altered mental state
3. **Raised lactate**

## Types of shock

Distributive shock presents differently

*250ml only if risk of fluid overload (renal/ heart failure, frail)

| Type of shock | Hypovolaemic | Cardiogenic | Obstructive | Distributive |
|---|---|---|---|---|
| **Causes** | **Reduced preload:**<br>• Haemorrhage<br>• Dehydration<br>• GI losses | **Pump failure:**<br>• Arrhythmias<br>• Cardiomyopathy<br>• MI | **Obstructed flow:**<br>• Tension pneumothorax<br>• Massive PE<br>• Aortic dissection<br>• Cardiac tamponade | **Reduced SVR:**<br>• Sepsis<br>• Anaphylaxis<br>• Neurogenic (SC injury above T6) |
| **Compensation** | ↑ SVR by vasoconc.<br>↑ **HR** | ↑ SVR by vasoconc.<br>(↑ contractility or HR) | ↑ SVR by vasoconc.<br>↑ **HR** | ↑ **HR**<br>↑ Contractility |
| **Clinical signs** | **Cool & mottled**<br>• Dehydration sunken eyes, ↓ turgor etc. | **Cool & clammy**<br>• Fluid overload if HF (↑ **JVP**, crackles, ↑ RR) | **Cool & clammy**<br>• Distended neck veins / raised JVP | **Warm & flushed**<br>• Fever if septic<br>• ↓ HR if neurogenic<br>• Rash if anaphylactic |
| | **General signs:** ↓ BP, ↑ CRT, ↓ UO, altered mental state (+ ↑ lactate) | | | |
| **Management** | High flow (15L) $O_2$ via non-rebreather mask<br>Early IV access<br>Treat cause | | | |
| | → **Fluid replacement**<br>Like-for-like (blood/ saline)<br>• 500ml bolus*<br>• Further volume given dependent on clinical effect | → **Depends on cause**<br>• ↑ HR = cardioversion<br>• ↓ HR = chronotropes<br>• ↓ contract = inotropes<br>**Avoid fluids** | → **Fluid therapy** (carefully)<br>→ **Remove obstruction**<br>• Tension pneumothorax = needle decompress & chest drain<br>• PE = thrombolysis<br>• Cardiac tamponade = pericardiocentesis<br>• Aortic dissection = surgery | → **Replace fluids**<br>→ **Vasopressors**<br>± **inotropes** |

## Management of shock[8]

**HDU/ICU admission if severe or refractory shock**

1. **Monitoring**
   - ABCDE assessment (regularly repeat)
   - Standard observations (RR, $SpO_2$, HR, BP, temp., AVPU)
   - Hourly urine output (insert catheter)
2. **Urgent investigations**
   - Blood gas (lactate, acid–base status)
   - Bloods (FBC, U&Es, CRP, coag screen, cultures, troponin/BNP)
   - ECG

**Advanced monitoring:**

- Central venous pressure (CVC)
- Invasive BP (arterial line)
- Continuous cardiac monitoring

**Other Ix:**

- Mast cell tryptase if suspect anaphylaxis
- X-match + G&S if haemorrhage

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[8] Frost PJ, Wise MP (2012) Early management of acutely ill ward patients. *BMJ*, 345:e5677

## Classifying haemorrhagic shock

| Class | Blood loss (ml) | Blood loss (%) | Pulse rate (/min) | Blood pressure | Resp rate (/min) | Urine output (ml/h) | CNS / mental state |
|---|---|---|---|---|---|---|---|
| I | <750 | <15 | <100 | Normal | 14–20 | >30 | Slightly anxious |
| II | 750–1500 | 15–30 | 100–120 | Normal | 20–30 | 20–30 | Mildly anxious |
| III | 1500–2000 | 30–40 | 120–140 | Decreased | 30–40 | 5–15 | Anxious/ confused |
| IV | >2000 | >40 | >140 | Decreased | >35 | Negligible | Confused/ lethargic |

NB: values differ at extremes of weight

Hypotension = late sign

## Dehydration & hypovolaemia

### RISK FACTORS:

**Reduced intake** – cognitive impairment e.g. dementia, children
**Increased loss** – exercise, hot environment, fever, vomiting, diarrhoea, diuretics, high output stoma

### SIGNS & SYMPTOMS:

| | Mild | Moderate | Severe |
|---|---|---|---|
| **Symptoms** | • Thirst<br>• Light-headedness<br>• Nausea | • Apathy/tiredness<br>• Dizziness, headache<br>• Muscle cramps | • Profound apathy & weakness<br>• Confusion → coma |
| **Signs** | • Possible postural hypotension | • Dry tongue<br>• Sunken eyes<br>• Reduced skin turgor<br>• Postural hypotension<br>• Tachycardia<br>• Oliguria | • Peripheral vasoconstriction<br>• Cold extremities<br>• Tachycardia<br>• Prolonged capillary refill time<br>• Oliguria/anuria<br>• Hypotension/shock |
| **Management**[9] | Encourage oral intake | Oral rehydration solution<br>IV fluids if cannot take fluids orally | IV fluids*:<br>**1.** Resuscitation bolus – reassess after bolus<br>**2.** Maintenance (total 25–30ml/kg/h) |

* IV rehydration should be as close to plasma as possible – a balanced crystalloid solution will reduce hyperchloraemic acidosis

*See the section on postoperative care in *Chapter 10: General surgical principles* for more information on fluid management

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[9] NICE CKS (2020) *Gastroenteritis* + NICE (2013) *Algorithms for IV fluid therapy in adults*

# Life-threatening arrhythmia

## Types of arrhythmia

- **Tachyarrhythmia** e.g. VF, VT, SVT, fast AF
- **Bradyarrhythmia** e.g. heartblock, sinus node dysfunction

## Management

1. **Commence continuous cardiac monitoring:** via an automated external defibrillator & obtain a 12-lead ECG
2. **Other investigations for cause:** FBC, U&Es, blood gas
3. **Management depends on type:**
    - If **no pulse** = cardiac arrest protocol (shockable & non-shockable rhythms)
    - If **pulse** but **unstable** = **synchronised** direct current cardioversion (DCC)
    - If **pulse** and **stable** = pharmacological Mx or temporary transcutaneous pacing (refractory bradycardia)

**Features of instability:**
1. Shock
2. Syncope
3. Myocardial infarction
4. Severe heart failure

### TREATMENT ALGORITHM FOR ADULT BRADYCARDIAS (WITH PULSE)[10]

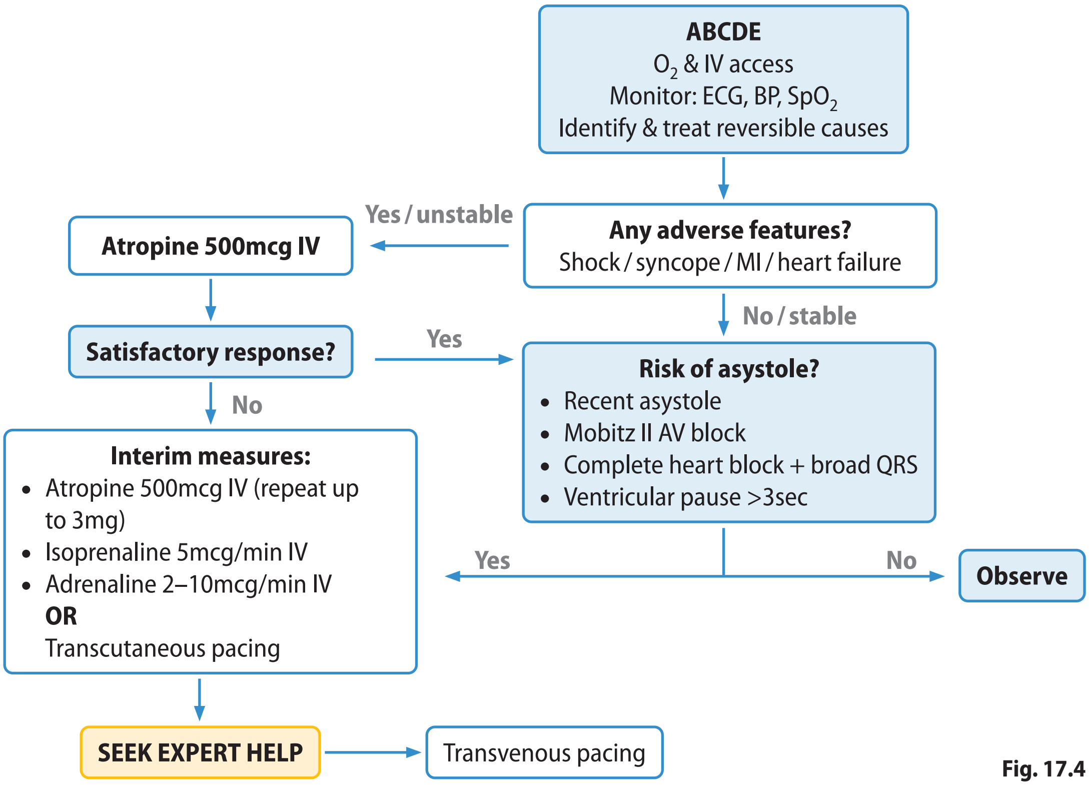
ABCDE
O₂ & IV access
Monitor: ECG, BP, SpO₂
Identify & treat reversible causes
Any adverse features?
Shock / syncope / MI / heart failure
Yes / unstable
Atropine 500mcg IV
Satisfactory response?
Yes
No / stable
Risk of asystole?
Recent asystole
Mobitz II AV block
Complete heart block + broad QRS
Ventricular pause >3sec
No
Interim measures:
Atropine 500mcg IV (repeat up to 3mg)
Isoprenaline 5mcg/min IV
Adrenaline 2–10mcg/min IV
OR
Transcutaneous pacing
Yes
No
Observe
SEEK EXPERT HELP
Transvenous pacing

**Fig. 17.4**

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[10] Resuscitation Council UK (2021) *Adult bradycardias*

## TREATMENT ALGORITHM FOR ADULT TACHYCARDIAS (WITH PULSE)[11]

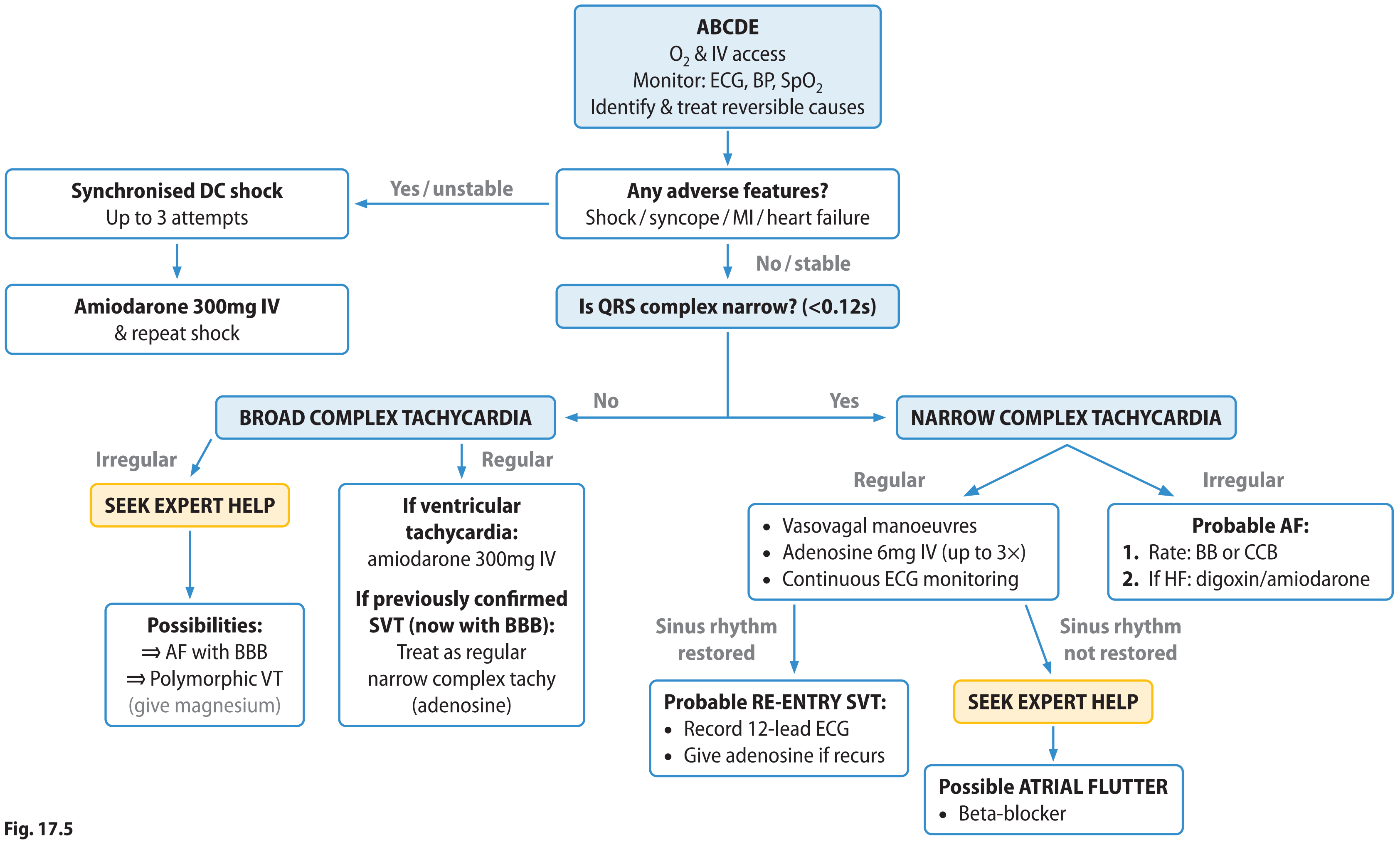
ABCDE
$O_2$ & IV access
Monitor: ECG, BP, $SpO_2$
Identify & treat reversible causes
Any adverse features?
Shock / syncope / MI / heart failure
Yes / unstable
Synchronised DC shock
Up to 3 attempts
Amiodarone 300mg IV
& repeat shock
No / stable
Is QRS complex narrow? (<0.12s)
No
Yes
BROAD COMPLEX TACHYCARDIA
Irregular
SEEK EXPERT HELP
Possibilities:
⇒ AF with BBB
⇒ Polymorphic VT
(give magnesium)
Regular
If ventricular tachycardia:
amiodarone 300mg IV
If previously confirmed SVT (now with BBB):
Treat as regular narrow complex tachy (adenosine)
NARROW COMPLEX TACHYCARDIA
Regular
• Vasovagal manoeuvres
• Adenosine 6mg IV (up to 3×)
• Continuous ECG monitoring
Sinus rhythm restored
Probable RE-ENTRY SVT:
• Record 12-lead ECG
• Give adenosine if recurs
Sinus rhythm not restored
SEEK EXPERT HELP
Possible ATRIAL FLUTTER
• Beta-blocker
Irregular
Probable AF:
1. Rate: BB or CCB
2. If HF: digoxin/amiodarone

**Fig. 17.5**

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[11] Resuscitation Council UK (2021) *Adult tachycardias*

# Cardiac arrest[12]

**Causes of cardiac arrest:** 4 Hs & 4 Ts

**H**ypoxia
**H**ypo-/hyperthermia
**H**ypo-/hyperkalaemia
**H**ypovolaemia
**T**ension pneumothorax
**T**amponade
**T**hrombosis
**T**oxins

**Rhythm check:**
**Shockable rhythms:** VF & pulseless VT
**Non-shockable rhythms:** asystole & pulseless electrical activity

**MINIMISE DISRUPTION TO CHEST COMPRESSIONS FOR RHYTHM CHECK & SHOCK**

**Post-arrest Ix:**

**A**BG + O$_2$
**B**loods + glucose
**C**XR
**D**ispose to ICU
**E**CG

**Mx of shockable rhythms:** VF & pVT

- **Shock ASAP**
- Continue chest compressions
- **Recheck cardiac rhythm every 2min**
- Re-administer shock if indicated

**After 3rd shock:**

- 1mg 1 in 10,000 IV adrenaline + 300mg amiodarone
- **Repeat adrenaline every 3–5min**

**Mx of non-shockable rhythms:** PEA & asystole

- **1mg IV adrenaline** (10ml 1:10,000)
- **Recheck cardiac rhythm every 2min**
- Repeat adrenaline every 3–5min

**If switches to shockable rhythm:**

- CPR until defib. is charged & ready to shock

## Adult basic life support (BLS)

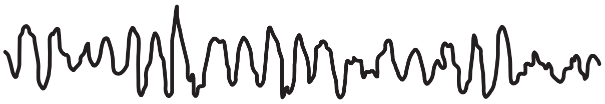
1. **Check for danger**
2. **Check for response**
3. **Call for help / pull alarm**
4. **Airway:** check for obstruction → head tilt, chin lift (or jaw thrust)
5. **Breathing:** look, listen, feel (10s)
6. **Circulation:** feel for carotid pulse (at same time as breathing assessment)
7. **Phone 999/2222**
8. **Start CPR:** 30 to 2 (100–120/min)
9. Continue until help arrives
10. **Attach automated external defibrillator (AED)**
    a) shout to stand back & remove O$_2$
    b) rhythm check
    c) shock if indicated
11. **Assess & treat underlying cause**
12. **Post-arrest care:** O$_2$ (94–98% sats) + temperature control no higher than 37°C

## Paediatric BLS

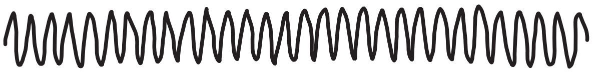
→ Head tilt, chin lift
→ **5 initial rescue breaths then** check for signs of life **including brachial pulse**
→ Compressions **15:2** (100–120/min)
→ Use finger or one hand if small

## Cardiac arrest rhythms

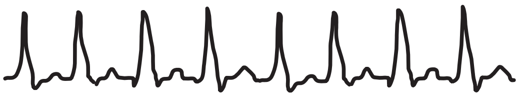
**VENTRICULAR FIBRILLATION:**



**PULSELESS VENTRICULAR TACHYCARDIA:**



**PULSELESS ELECTRICAL ACTIVITY:**



**ASYSTOLE:**

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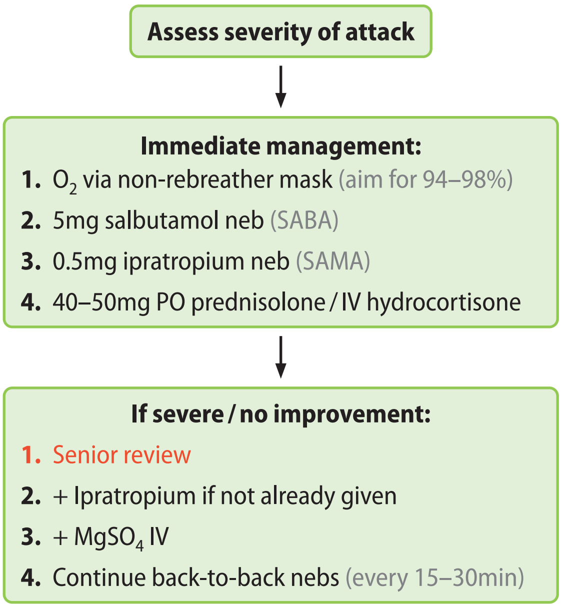
[12] Resuscitation Council UK (2021) *Adult advanced life support guidelines*

# Specific presentations

## Acute asthma[13]



**Assess severity of attack**

**Immediate management:**

1. $O_2$ via non-rebreather mask (aim for 94–98%)
2. 5mg salbutamol neb (SABA)
3. 0.5mg ipratropium neb (SAMA)
4. 40–50mg PO prednisolone / IV hydrocortisone

**If severe / no improvement:**

1. Senior review
2. + Ipratropium if not already given
3. + $MgSO_4$ IV
4. Continue back-to-back nebs (every 15–30min)

**Fig. 17.6**

| Severe attack | Life-threatening attack |
|---|---|
| Incomplete sentences | Exhaustion/confusion |
| Accessory muscles | Silent chest |
| Hyperinflated chest | Cyanosis |
| Pulsus paradoxus* | PEF <33% of best |
| PEF 33–50% of best | $SpO_2$ <92% |
| RR >25 | ↓HR & ↓BP |
| HR >110 | **ABG:** ↑$CO_2$, $O_2$ <8, low pH |
| *↓ SBP with inspiration | |

### MANAGEMENT:

**O**xygen (15L/min)
**S**albutamol (5–10mg neb)
**H**ydrocortisone (100mg IV) **or** prednisolone (50mg PO)
**I**pratropium (500microgram neb)
**T**heophylline (PO) [aminophylline = IV form]
**M**agnesium sulphate (IV)
**E**scalate!

Purple steps are for severe cases

## Acute COPD exacerbation[14]

### SIGNS:

1. Altered sputum volume/colour
2. Reduced exercise tolerance
3. Fever/malaise/lethargy

### INVESTIGATIONS:

- Sputum sample if purulent
- ABG = T2RF (respiratory acidosis)
- FBC, U&Es
- CXR, ECG

### MANAGEMENT:

1. **Nebulised bronchodilators** (back-to-back): SABA & SAMA + theophylline if insufficient response
2. **Oral corticosteroids:** 30mg OD prednisolone (5d)
3. **Oxygen:** **88–92%** via Venturi mask if $CO_2$ retainer (92–96% if not)
4. **Antibiotics:** e.g. doxycycline (5–7d) if ≥2 of ↑sputum, ↑purulence, ↑dyspnoea
5. **Refer to respiratory team:** consider NIV if persistent T2RF

---

[13] SIGN (2019) *British guideline on the management of asthma* [158]
[14] NICE (2018, updated 2019) *Chronic obstructive pulmonary disease in over 16s* [NG115]

## Acute coronary syndrome[15]

### MANAGEMENT:

| | | |
|---|---|---|
| **M** | Morphine + metoclopramide | 5–10mg |
| **O** | Oxygen | Target 92–96% sats |
| **N** | Nitrates | GTN spray (SL) or IV nitrate |
| **A** | **Antiplatelets**<br>**1.** Aspirin +<br>**2.** Clopidogrel/ticagrelor | **1.** 300mg chewable<br>**2.** Depends on Trust guidelines |

→ **If STEMI = PCI within 120min or thrombolysis**

(see *Chapter 1: Cardiology* for further details)

## Anaphylaxis[16]

### INITIAL MANAGEMENT:

**1 in 1000 = FATAL**

1. **ABCDE** (and call for help!)
   - **Airway:** secure airway
   - **Breathing:** 15L $O_2$ via NRM + SABA if wheezy
   - **Circulation:** lie supine with legs raised
2. **IM adrenaline 1:1000**
   - ≤6m = 100–150mcg (0.10–0.15ml)
   - <6y = 150mcg (0.15ml)
   - 6–12y = 300mcg (0.30ml)
   - >12y = 500mcg (0.50ml)

→ repeat in 5min if no improvement

Hydrocortisone no longer given in routine emergency treatment

3. **Further treatment options**
   - **If stable:** non-sedating antihistamine e.g. cetirizine
   - **If refractory/shock:** IV fluid bolus + IV adrenaline infusion

### FURTHER MANAGEMENT:

- **Monitor** – pulse oximetry, ECG, BP (for at least 6h)
- **Serum tryptase:** on arrival then 1–4h later (↓s after 4h) + 24h after resolution
- **Prednisolone PO:** 3–5d
- **± EpiPen prescription** & education
- **Follow-up:** with GP in 3–5d
- **Allergy clinic referral**
- **Document in notes**

---

[15] NICE (2020) *Acute coronary syndromes* [NG185]

[16] Resuscitation Council UK (2021) *Anaphylaxis guidance*

## Heatstroke

**Heatstroke** = core body temperature >40°C with CNS dysfunction
**Heat exhaustion** is a milder form with core body temperature 37–40°C

### SYMPTOMS:

- Core body temperature >40°C
- CNS dysfunction (confusion, coma, seizures)
- Intense thirst
- Weakness, dizziness, syncope

### INVESTIGATIONS:

- 12-lead ECG
- FBC, U&Es, LFTs, TFTs, clotting screen, blood glucose
- Urinalysis & CK – rhabdomyolysis
- VBG/ABG – mixed respiratory & metabolic acidosis from hypermetabolic state

### MANAGEMENT[17]: ABCDE

1. **Rapid active cooling** – wetted cloths, ice packs, cold/ice water immersion
2. **Secure airway if compromised** + supplemental humidified oxygen if not maintaining saturations
3. **Further cooling** – cool IV fluids
    - If exertional, consider 3% sodium chloride for associated hyponatraemia

**Monitoring:** core temperature, haemodynamic status, vital signs, blood gases/pH

**For less severe heat exhaustion:** removing excess clothing, moving to a cooler environment & giving oral isotonic fluids is usually sufficient

## Hypothermia

**Hypothermia** = core body temperature <35°C

### SYMPTOMS:

- Core body temperature <35°C
- Cognitive impairment
- Shivering
- Tachypnoea → respiratory depression → apnoea
- Tachycardia → bradycardia
- Cold-induced diuresis

Rectal/oesophageal temperature probe recommended for accurate measure of core body temperature

### INVESTIGATIONS:

- 12-lead ECG
- U&Es, blood glucose, clotting screen, TFTs, CK (if long lie)
- VBG/ABG – mixed respiratory & metabolic acidosis
- CXR – may show pulmonary infiltrates or oedema

### MANAGEMENT[18]: ABCDE – severe hypothermia needs urgent critical care support

1. **Stop further cooling** – remove from cold environment, remove cold/wet clothing
2. **Secure airway if compromised** + supplemental humidified oxygen if not maintaining saturations
3. **Re-warming** – depends on severity
    - **Mild/moderate** – passive warming: blankets / forced air warmer, warmed IV fluids
    - **Severe** – active warming: bladder lavage, intravascular/internal ECLS

**Aim to increase temperature at rate of 0.5–2°C per hour**

**ECLS: extra-corporeal life support** preferred method if in cardiac arrest → needs careful monitoring

**Monitoring:** core temperature, haemodynamic status, vital signs, blood gases/pH

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[17] BMJ Best Practice (2021) *Heat stroke*
[18] BMJ Best Practice (2021) *Hypothermia*

# 18

# RHEUMATOLOGY

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## ABBREVIATIONS

**Abs** – Antibodies
**ACL** – Anterior cruciate ligament
**ADLs** – Activities of daily living
**AkS** – Ankylosing spondylitis
**ALP** – Alkaline phosphatase
**ANA** – Antinuclear antibody
**ANCA** – Antineutrophil cytoplasmic antibody
**Anti-CCP** – Anticyclic citrullinated peptide
**Anti-SM** – Anti-smooth muscle
**APL** – Abductor pollicis longus
**APS** – Antiphospholipid syndrome
**BASDAI** – Bath Ankylosing Spondylitis Disease Activity Index
**BASFI** – Bath Ankylosing Spondylitis Functional Index
**CBT** – Cognitive behavioural therapy
**CCB** – Calcium channel blocker
**CK** – Creatine kinase
**CNS** – Central nervous system
**COCP** – Combined oral contraceptive pill
**CPPD** – Calcium pyrophosphate crystal deposition
**CTD** – Connective tissue disease
**CTS** – Carpal tunnel syndrome
**DAS** – Disease activity score
**DIPJ** – Distal interphalangeal joint
**Dm** – Dermatomyositis
**DMARD** – Disease-modifying antirheumatic drug
**DRE** – Digital rectal examination
**dsDNA** – Double-stranded DNA
**EBV** – Epstein–Barr virus
**EPB** – Extensor pollicis brevis
**EPL** – Extensor pollicis longus
**FDP** – Flexor digitorum profundus
**FDS** – Flexor digitorum superficialis
**FPL** – Flexor pollicis longus
**GCA** – Giant cell arteritis
**GTPS** – Greater trochanteric pain syndrome
**HSP** – Henoch–Schönlein purpura
**IA** – Intra-articular
**IBD** – Inflammatory bowel disease
**IBS** – Irritable bowel syndrome
**IIM** – Idiopathic inflammatory myopathies
**LMN** – Lower motor neurone
**MCL** – Medial collateral ligament
**MCPJ** – Metacarpophalangeal joint
**MS** – Multiple sclerosis
**MSK** – Musculoskeletal
**MSUM** – Monosodium urate monohydrate
**MTPJ** – Metatarsophalangeal joint
**MTX** – Methotrexate
**OA** – Osteoarthritis
**OT** – Occupational therapy
**PCL** – Posterior cruciate ligament
**PIPJ** – Proximal interphalangeal joint
**Pm** – Polymyositis
**PMR** – Polymyalgia rheumatica
**PPI** – Proton pump inhibitor
**PsA** – Psoriatic arthritis
**PUD** – Peptic ulcer disease
**RA** – Rheumatoid arthritis
**RF** – Rheumatoid factor
**RNP** – Ribonucleoprotein
**ROM** – Range of movement
**RTA** – Road traffic accident
**SIJ** – Sacroiliac joint
**SLE** – Systemic lupus erythematosus
**SPF** – Sun protection factor
**STI** – Sexually transmitted infection
**TB** – Tuberculosis
**TCA** – Tricyclic antidepressant
**TGs** – Triglycerides
**UMN** – Upper motor neurone

# Regional periarticular pain

**Definitions:**

**Enthesopathy:** disorder of tendon/ligament/joint capsule
**Tendinitis:** tendon inflammation
**Tenosynovitis:** tendon sheath inflammation
**Bursitis:** inflammation of bursae
NB: some muscles insert directly into bone

## Overview of musculoskeletal injuries

**Causes:** due to direct or indirect trauma

- **Contusion:** direct blow / compressive force to *the muscle*
- **Strain:** excessive stretching of the muscles causes microtrauma at the *musculotendinous junction*
- **Sprain:** excessive stretching/force causing rupture of collagen bundles within a *ligament*

**General approach:**

1. **History:** mechanism of injury, onset and characteristic of symptoms, previous episodes
2. **Examination:** asymmetry, deformity, tenderness, bruising, ROM
3. **Imaging:** consider X-ray if suspicious of fracture, consider MRI if ongoing pain despite treatment / complex injury
   → often no imaging required to make diagnosis

## Strain & overuse injuries

*→ repeated micro-trauma to muscle/tendon fibres*

**Risk factors for strain injuries:**

- Age = weaker tendons & longer to heal
- Imbalances in strength/flexibility
- Anatomical abnormalities e.g. flat feet
- Previous injuries
- Poor technique / inappropriate footwear
- Training errors (e.g. over-training)

### CLINICAL FEATURES:

- **Exact time/mechanism of injury** may be identified in muscle strains
- **Pain during, or within 12h of activity**
- Pain = non-progressive
- No systemic Sx

**On examination:**

- **Localised** periarticular pain (along path of affected structure)
- Pain **resisting active movements** (passive > active movement)
- No/mild visible inflammation

### GENERAL MANAGEMENT OPTIONS:

- Analgesics e.g. NSAIDs
- Rest, compression, ice/heat
- Supports/adjuncts
- Physiotherapy
- Steroids (PO or injections)
- Surgery
- Lifestyle modifications

**Lifestyle changes**
→ modify work/exercise
→ warm up / cool down
→ improve technique

## Epicondylitis

### CLINICAL FEATURES:

- **Gradual onset**
- Localised epicondyle pain (worse with use, eased with rest)
- Normal passive ROM

### MANAGEMENT: stop the exacerbating action

**Conservative:** NSAIDs, physio, splints/adjuncts
**Medical:** steroid injection if Tx-resistant
**Surgical:** release incision if Tx-resistant

| | **Lateral epicondylitis ('tennis elbow')**<br>= inflammation of **common extensor tendon** | **Medial epicondylitis ('golfer's elbow')**<br>= inflammation of **common flexor-pronator tendon & ulnar collateral ligament** |
|---|---|---|
| **Peak age** | 40–55y (more common) | 50–60y (less common) |
| **Risk factors** | Obesity, smoking, carpal tunnel, other tendinopathies<br>After sudden unaccustomed activity<br>(e.g. carry heavy objects → then report pain) | Manual work, sports |
| **Signs O/E** | • Tenderness over lateral epicondyle<br>• Pain with **resisted wrist extension** (Cozen's test)<br>• Pain with **resisted wrist supination**<br>Patients report pain gripping & lifting heavy items | • Tenderness over medial epicondyle<br>• Pain with resisted wrist flexion & pronation<br>± **cubital tunnel signs** (tingling/numbness) |

## De Quervain's tenosynovitis

→ ***inflammation of synovial sheaths of thumb tendons*** *(EPB & APL)*

**SYMPTOMS:**

- Pain on radial border of wrist/forearm (worse with thumb flexion + ulnar deviation → **Finkelstein's test**)
- Swelling around styloid process of radius

**MANAGEMENT:**

- Rest, splinting & NSAIDs
- Local corticosteroid injections
- Surgical release of tendon tunnel if Sx persist

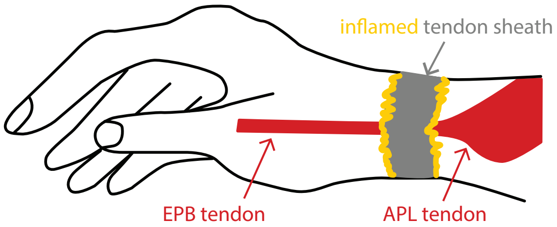


**Fig. 18.1** De Quervain's.

## Rotator cuff lesions

| | TENDON TEARS | CALCIFIC TENDINITIS | ADHESIVE CAPSULITIS (FROZEN SHOULDER) |
|---|---|---|---|
| **Pathogenesis** | **Injury or progressive degeneration** with age/use | Deposits of calcium hydroxyapatite crystals<br>**(30–55y F>M)** | Capsular fibrosis seen in **middle-aged/elderly** |
| **Symptoms** | **Weakness & pain** on shoulder movement<br>(may also disturb sleep) | **Very severe shoulder pain** & stiffness<br>± brachial plexus neuritis | Shoulder **pain, followed by increasing stiffness**<br>(no swelling or crepitus) |
| **Signs O/E** | Reduced ROM **active** > **passive** in direction of muscle action<br>(If pain with movement but not weakness suggests tendinitis, not tear yet) | Loss of ROM **(active & passive)** | **Freezing** (pain) → **frozen** (stiffness) → **thawing** (resolves)<br>Loss of ROM **(active = passive)** esp. external rotation <50%<br>**Normal strength** |
| **Investigation & diagnosis** | • Hx & exam<br>• USS/MRI<br>• X-ray (r/o arthritis / subacromial bursitis)<br>Only refer for imaging/2° care if no improvement in 12w or traumatic injury | • Hx & exam<br>• X-ray (& USS) | • Hx & exam<br>• X-ray (& USS)<br>= global loss of movement with no abnormal radiography |
| **Management** | • Rest, ice, analgesia (NSAIDs)<br>• Steroid injections (with caution)<br>• Physiotherapy<br>• Arthroscopic/open surgery<br>► if traumatic tear<br>► if high functional demand<br>► if no improvement in 12w | • Rest & analgesia (NSAIDs)<br>• Steroid injections<br>• Arthroscopic incision if Sx don't improve<br>+ education on how to adapt to live with reduced ROM | • Analgesia (NSAIDs) ± steroid injections (but not inflammatory so less useful)<br>• Physiotherapy<br>• Arthroscopic release if no improvement in 12w<br>COUNSEL: self-limiting in 1.5–2y |

Common 35–75y

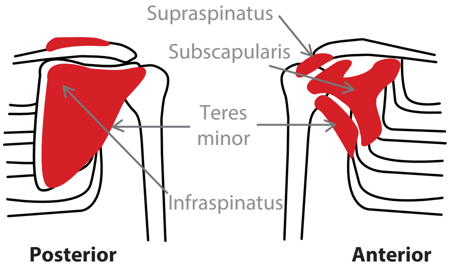


**Fig. 18.2** Rotator cuff muscles.

**Risk factors for frozen shoulder:**

- Age (>40y) & female
- Following injury/surgery
- Shoulder immobility
- DM, thyroid disease, CVD, HTN

## Subacromial bursitis & impingement

→ **Supraspinatus tendon** runs through narrow space between head of humerus & underside of acromion
→ On shoulder abduction, **subacromial bursa** normally prevents friction between tendon & acromion

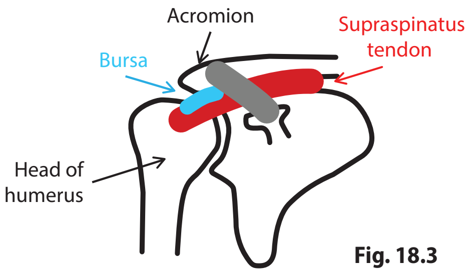


**Fig. 18.3**

### SYMPTOMS OF IMPINGEMENT SYNDROME:

- Pain on shoulder **abduction between 45 and 120°** (mid-arc) (difficulty with overhead tasks)
- Pain may be present at night / disrupt sleep
- ± Arm weakness

→ **O/E:** reduced **active** movement > passive
→ Pain with **Hawkins test & 'painful arc test'**
→ 'Empty can' test negative (distinguishes from supraspinatus tendinitis)

### CAUSES OF IMPINGEMENT SYNDROME:

1. **Subacromial bursitis** – inflamed & swollen so narrows space (injury/overuse)
2. **Supraspinatus tendinitis** – inflamed & thickened tendon (injury/overuse)
3. **Acromioclavicular arthritis** – bony spurs narrow joint space (age)

### MANAGEMENT:

**Conservative:** NSAIDs, physio
**Medical:** steroid injection
**Surgical:** remove bony tissue

*Risk factors for biceps tendon rupture: smoking, steroid use, elderly*

## Biceps tendon rupture

### SYMPTOMS:

- Sudden pop on heavy lifting (if elderly may be with minor activity)
- 'Popeye' appearance

*= usually older patients when lifting something heavy*

### MANAGEMENT:

- **If younger / fit & active:** surgery
- **If older / not fit & active:** leave for self-resolution → rest, ice, compression, elevation

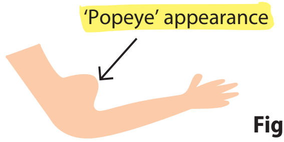


**Fig. 18.4**

## Greater trochanter pain syndrome (GTPS)

### SYMPTOMS: 50% = bilateral

- Pain over thigh/buttock (worse when lying on side & during exercise)
- Localised tenderness to touch
- **No difficulty putting on shoes/socks (r/o OA of hip)**

**ON EXAMINATION:** point tenderness over greater trochanter

**INVESTIGATION:** Hx & exam, X-ray, MRI

### CAUSES:

- Gluteal tendinitis / enthesitis
- Trochanteric bursitis: self-limiting inflammation of bursa

### MANAGEMENT: >2/3 resolve in 1y with this Mx

**Conservative:** NSAIDs, physio & rest
**Medical:** steroid injection

**Risk factors for GTPS:**

- Age (>40y) & **female**
- Obesity
- **Total hip replacement surgery**
- Injury/ trauma e.g. fall on that side
- Prolonged sitting / weight bearing
- Repetitive use damage (long walking / running distances)

**Differentials of GTPS:**

- Hip OA
- Labral tear
- Inflammatory joint disease
- Referral of lumbar pain
- Neck of femur fracture

## Patellar bursitis

**CAUSE:** prolonged kneeling

**SYMPTOMS:** localised anterior knee pain & tenderness + fluctuant swelling

**MANAGEMENT:** avoid kneeling (incision & drainage if infected)

→ **Prepatellar:** 'housemaid's knee'
→ **Infrapatellar:** 'clergyman's knee'

## Achilles tendinitis

→ *overuse injury (most common in long distance runners)*

**SYMPTOMS:**

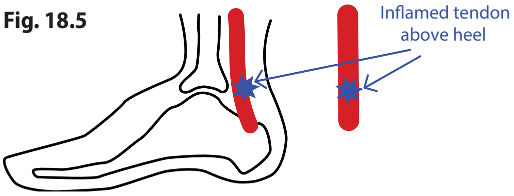
- Ache/pain above heel after running / climbing stairs
- Morning stiffness

**MANAGEMENT:**

- Rest, ice & analgesia (NSAIDs)
- Stretching calf muscles & physio

NB: can be associated with inflammatory arthropathies

Fig. 18.5



**Risk factors for Achilles tendinitis:**

- Age & male gender
- Flat feet / poor footwear
- Long distance running

*middle-aged just doing sport once a week*

**Achilles enthesitis**

= Inflammation of Achilles tendon at **insertion point & terminal tendon fibres**

→ Pain may radiate down under heel

## Plantar fasciitis

→ *overuse injury causing inflammation of **plantar fascia***

**SYMPTOMS:**

- Stabbing pain under heel (worse with first steps after prolonged resting)
- Worsened with prolonged standing / exercise

**ON EXAMINATION:**

- **Highly localised** tenderness at medial tuberosity of calcaneus
- Pain exacerbated with **active or passive toe dorsiflexion**

**MANAGEMENT:** resolves in 6–8w

- Rest, ice & analgesia (NSAIDs)
- Physio/stretching
- **Lifestyle:** lose weight, change footwear

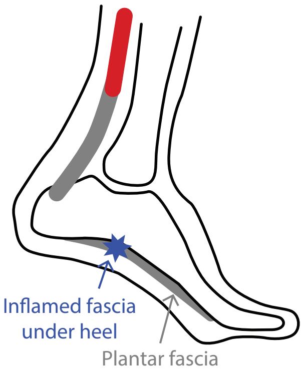


Fig. 18.6

**Risk factors for plantar fasciitis:**

- Prolonged standing / running
- Poor trainers / hard ground
- Flat feet
- Obesity
- Middle-aged & female

**DDx of plantar fasciitis:** inflammatory conditions, calcaneal stress fracture, nerve entrapment

## Subcalcaneal bursitis

→ *inflammation of bursa between calcaneus & plantar fascia*

**SYMPTOMS:**

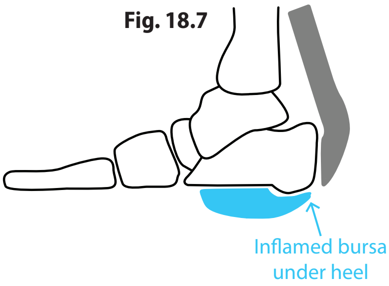
- Dull ache under heel (worsens throughout day)

**MANAGEMENT:** resolves in 6–8w

- Rest, ice & analgesia (NSAIDs)
- Comfortable footwear, orthoptics

Same risk factors as for plantar fasciitis

Fig. 18.7



**Grading of sprains**

**Grade 1: mild** – microscopic tears in ligament
**Grade 2: moderate** – partial tearing of ligament
**Grade 3: severe** – complete tear of the ligament

**Ottawa rules**[1]

**Ottawa ankle rules** to rule out fracture

**Only X-ray ankle if:**
Pain near the malleolus
**and either**
Unable to weight bear (4 steps) immediately after injury & when in emergency department
**or**
Bony tenderness at posterior edge or tip of malleolus

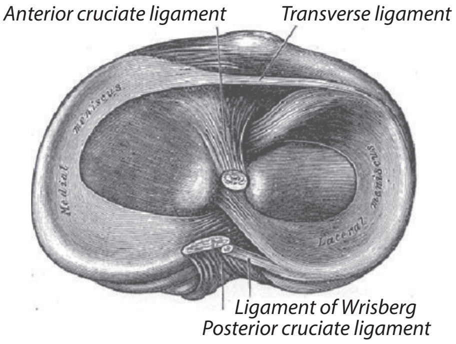


**Fig. 18.8** Anatomy of knee joint.

→ **ACL injuries = most common (50%)**
→ **Second most common = MCL injuries**
→ Often **multiple ligaments** are involved

**Grading instability**

| Grade | Injury | Exam finding |
|---|---|---|
| **0** | Normal | **Normal** – firm end-point |
| **1** | Sprain | **<5mm** translation ↑ |
| **2** | Partial tear | **5–10mm** ↑ translation |
| **3** | Complete tear | **>10mm** ↑ translation |

## Ankle sprain

### SYMPTOMS:

- Pain, swelling, bruising around the ankle

### MANAGEMENT: resolves in 6–8w

- Rest, ice, compression & analgesia (NSAIDs)
- Early mobilisation & strengthening exercises

Use Ottawa ankle rules to decide if X-ray is indicated

## Meniscal injuries of the knee

- Menisci act to spread the load & disperse friction
- Medial meniscus more commonly damaged & associated with ACL tears
- Inner 75% of menisci ('white zone') = **avascular so poor healing**

### CAUSES OF MENISCAL DAMAGE:

- Degenerative tears
- Acute tears → twisting injury

### DIAGNOSIS:

- Hx & examination: McMurray's test
- X-ray to r/o fracture/OA
- MRI scan = **diagnostic**

### SYMPTOMS: ± swelling

- **Pain:** intermittent & on knee movement
- **Mechanical Sx:** locking & giving way

### MANAGEMENT:

- **Arthroscopic repair** (if Sx are serious / younger patient)
- **Conservative Tx** (if degenerative tear / OA) → activity modification, physio, NSAIDs

## Ligamentous injuries of the knee

### CAUSES OF LIGAMENTAL DAMAGE:

- **ACL/PCL injuries:** rotational injuries when foot is planted e.g. footballers, skiers
- **Isolated PCL injury:** RTA ('dashboard injury')
- **Collateral ligament injuries:** lateral impact / opposing forces at knee & ankle

→ common in athletes / people playing sports

### SYMPTOMS: pain → may hear 'pop' at time of injury

- **ACL/PCL:** acute swelling (30 min) → have to stop activity / may be unable to weight bear
- **Collateral ligament:** more insidious swelling / Sx onset (24h)

### DIAGNOSIS:

- **Hx & examination:** grade instability 0–3 (anterior & posterior drawer, Lachman's, varus/valgus force)
- **X-ray:**
    - r/o fracture & OA
    - fat-fluid level suggests bone injury (lipohaemarthrosis)
- **MRI**

### MANAGEMENT:

- **ACL/PCL:** replacement with hamstring or patellar tendon graft → don't tend to heal on their own
- **Collateral ligaments:** immobilisation with knee brace & physio

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[1] Stiell I (1996) Ottawa Ankle Rules. *Can Fam Physician*, 42:478

# Neck & back pain

**Red flags: need urgent MRI**

- New onset in <20y or >55y
- Constant night pain
- Progressive motor weakness
- Thoracic back pain
- Saddle anaesthesia
- Bladder/bowel incontinence
- Hx of trauma/cancer
- Unexplained weight loss
- Fever
- Steroid use
- Recent infection

## Differentials of neck/back pain

| Structural | Inflammatory | Destructive | Metabolic | Referred pain |
|---|---|---|---|---|
| • Mechanical<br>• Disc prolapse<br>• Spinal stenosis<br>• Spondylolisthesis | • Spondyloarthropathies<br>• Sacroiliitis<br>• Polymyalgia rheumatica | • Malignancy (1° or 2°)<br>• Infection (discitis, osteomyelitis, TB) | • Osteoporosis<br>• Osteomalacia<br>• Paget's | • Major viscera<br>• Uro-genitary<br>• Aorta<br>• Hip |

## Cervical radiculopathies

**CERVICAL SPONDYLOSIS** → cervical radiculopathy caused by age-related degenerative changes to spine

- Ageing causes **disc degeneration** (dehydration & flattening) = ↓ shock absorption
- Results in bony changes of vertebrae → **osteophyte development**
- Osteophytes **'pinch' nerve roots** as they leave spinal canal
- **May develop into myelopathy**

**Symptoms:**

- Pain in neck (radiating down arm as a 'dull ache'/'toothache') → **'brachial neuralgia'**
- Tingling/numbness in one dermatome
- ± Weakness in one arm

**On examination:**

- Pain reproduced with **lateral neck flexion towards affected side** ↳ 'Spurling sign'
- **Motor signs:** modest upper muscle weakness
- **Sensory signs:** reduced pin-prick sensation discrimination (in one dermatome)
- **LMN signs:** hyporeflexia, hypotonia

**Diagnosis:** usually clinical

→ **Neurological examination:** myotomes, dermatomes, reflexes
→ **MRI** if no improvement / considering surgery

**Management:** mostly self-limiting in 6–12w

1. **Conservative:**
    - Rest, physiotherapy, analgesia (NSAIDs or neuropathic – TCAs)
    - Hard collar for neck immobilisation
2. **Surgical:**
    - ACDF (anterior cervical discectomy & fusion) if persistent/worsening/severe Sx

**CERVICAL DISC PROLAPSE** = seen in those aged 30–40y; usually Hx of mild neck trauma

**Symptoms:**

- Pain in neck
- Tingling/numbness/paraesthesia in one dermatome of arm
- ± weakness in one arm

**Management:**

1. **Conservative:** rest, physiotherapy, analgesia (NSAIDs or neuropathic – TCAs)
2. **Surgical:** microdiscectomy

**Investigation:**

→ **Neurological examination:** myotomes, dermatomes, reflexes
→ **MRI** → recommended for cervical spine (assess need for surgery)

**Radiculopathies** = conditions where 'pinched' **nerve roots** cause **pain, paraesthesia, weakness** in a **dermatomal distribution (unilateral)**
→ **LMN signs** (hyporeflexia, hypotonia)

**Myelopathies** = conditions where compressed **spinal cord** causes **pain, paraesthesia, weakness bilaterally** + other neurological symptoms
→ **UMN signs** (hyperreflexia, hypertonia, spasticity)

| **Upper limb dermatomes** | |
|---|---|
| **C5** | 'Regimental **badge area**' |
| **C6** | **Thumb** & index finger |
| **C7** | **Middle finger** |

## Cervical myelopathy

**CERVICAL CANAL STENOSIS** → cord compression = more serious than cervical radiculopathy

**Causes:**

- **Age-related degeneration:** osteophyte formation & ligament hypertrophy
- **Disc bulging/herniation:** consider in younger patients

Affects middle-aged/elderly

**Symptoms:** gradual onset so late presentation & Dx

- **Gait abnormalities:** spastic & ataxic
- **Loss of fine motor skills** → difficulty fastening buttons, writing etc.
- Later = tingling in fingers (may be misdiagnosed as carpal tunnel syndrome) ± sphincter dysfunction

**On examination:**

- **Gait abnormalities:** spastic & ataxic
- Wasting on shoulder girdle muscles
- UMN signs: spasticity, clonus, +ve Hoffman's/Babinski, hyperreflexia, hypertonia

**Investigations:** X-ray & MRI

**Management:**

**Surgical intervention:** laminectomy → recommended as progressive deterioration

**Hoffman's sign:** flick middle finger & watch for reflexive movement of index/thumb

→ **positive in UMN pathology** e.g. spinal cord compression

## Lumbar radiculopathies

**LUMBAR DISC PROLAPSE** = common in those aged 25–55y

**Pathology:**

- With age = increased risk of prolapse through defect in surrounding annulus fibrosus
- Results in **compression of nerve roots**

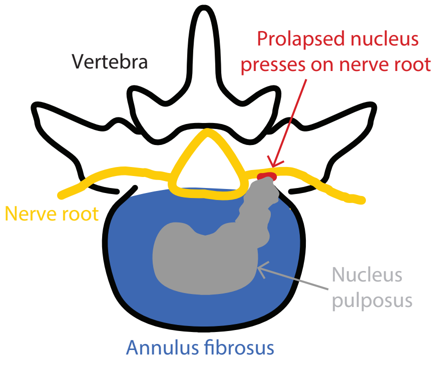

**Fig. 18.9** Anatomy of disc prolapse.

**Symptoms:** often onset during lifting/bending/twisting

- Stabbing lower back pain
  → radiating down the leg/buttock
- Numbness/tingling (in one leg)

**On examination:**

- Pain reproduced with **straight leg raise**
- **Motor signs:** modest lower muscle weakness (usually unilateral)
- **Sensory signs:** reduced pin-prick sensation discrimination (one dermatome)
- **LMN signs:** hyporeflexia, hypotonia
- May be **scoliosis** due to paravertebral muscle spasm

**Diagnosis:** usually clinical

- **Neurological examination:** myotomes, dermatomes, reflexes
- **MRI** if no improvement / considering surgery

**Management:** 90% self-limiting in 6–12w

1. **Conservative:** rest, physiotherapy, analgesia (NSAIDs or neuropathic – TCAs)
2. **Surgical:** if no improvement in 6w
   - Nerve root block (under fluoroscopic guidance)
   - Microdiscectomy (remove piece of prolapsed disc)

| Lower limb dermatomes | |
|---|---|
| **L4** | **Inner shin**, below knee |
| **L5** | **Buttock**, lateral calf, big toe |
| **S1** | Post. thigh & **calf**, little toe |

Usually **posterolateral** herniation of disc, therefore compresses **nerve root below** i.e. herniated L5/S1 disc will compress S1 nerve root

**Sciatica** = pain along path of sciatic nerve (radiates down to toe) due to disc prolapse, stenosis or osteophytes affecting **nerve roots L4–S1**

Discs are named after vertebra above & below e.g. L4/5 or L3/4

**Complications of surgery:**
Nerve damage, CSF leak, infection, haemorrhage, back pain

**CAUDA EQUINA SYNDROME** → compression of cauda equina nerve roots causing pain, paraesthesia & weakness

**Symptoms: usually bilateral & acute onset**
- Stabbing lower back & leg pain
- Lower limb weakness
- Bladder/bowel disturbance
- Saddle anaesthesia
- Sexual dysfunction

**Investigations:**
- Lower limb motor function
- Lower limb sensory function
  → esp. perianal (S3, S4, S5)
- Reflexes
- **DRE → sphincter tone**
- Pre- & post-micturition bladder scan (>200ml)
- **MRI** – may show disc prolapse

**Causes:**
- **Herniated lumbar disc**
  **= most common**
- Spinal stenosis
- Spinal tumour
- Spinal infection (abscess, TB)
- Severe injury to lower back
- Congenital malformation

**Management:**
**Immediate referral to neurosurgery**
→ urgent decompression e.g. via discectomy

## Lumbar myelopathy

**LUMBAR CANAL STENOSIS → cauda equina compression** causing **neurogenic claudication**

Usually L4/5 or L5/S1 level

**Causes:**
1. **Age-related degeneration:** osteophyte formation & ligament hypertrophy
2. **Disc bulging/herniation:** consider in younger patients

**Symptoms:**
- Hx of back pain → dermatomal distribution
- Leg pain / weakness / heaviness that limits walking distance → distance progressively decreases
  → **slowly** relieved by rest
  → feels better bending forwards / going uphill

**Investigations:**
1. **History & examination**
2. **Imaging:** X-ray + CT/MRI

**Management:** conservative
(lumbar laminectomy if severe)

- Lumbar canal stenosis affects **middle-aged/ elderly**
- May be 1st presentation of prostate cancer due to spinal mets

**DDx:** vascular claudication

| Intermittent claudication | Spinal stenosis |
|---|---|
| **Fixed** claudication distance | **Variable** claudication distance |
| **Worse** walking **uphill** | **Better** walking **uphill** |
| Pain relieved after 1–2min rest | Pain disappears after 15–30min |
| **O/E:** absent peripheral pulses | **O/E:** pulses present but neuro signs |

**LUMBAR SPONDYLOSIS** → age-related degeneration (see *Cervical spondylosis*)

**SPONDYLOLYSIS** → stress fractures in 'pars interarticularis' of vertebra (most commonly L5)

**Causes:** often young athletes

**Symptoms:** often asymptomatic (unless causing spondylolisthesis)

**Management:**
1. **Conservative:** rest, physio, NSAIDs, bracing
2. **Surgery:** if serious slip / worsening Sx

**SPONDYLOLISTHESIS** → slippage of one vertebra on the one below
**Causes:** spondylolysis, age-related degeneration
**Symptoms:** neck/back pain + radiculopathy (pain, numbness, tingling)

## Other causes of neck & back pain

**MECHANICAL BACK PAIN:** = very common (90% of back pain)!

**Cause:** many possibilities → joints, tendons, ligaments, discs, bones

**Symptoms:**

- Worse with movement, relieved with rest
- Sudden onset & may be recurrent

± stiffness (<30min), muscular spasm, scoliosis

**Investigations:**

- **Consider X-ray/MRI if:**
  - >50y or <20y
  - Red flags of back pain or suspect malignancy
  - Suspect inflammatory cause e.g. ankylosing spondylitis

**Management[2]:** 80% self-limiting in 6w

Key part of Mx

→ **Reassure & educate:** continue normal activity

→ **Exercise & physiotherapy:** encourage activity, good posture → yoga/ Pilates/swim/walk

→ **Analgesia:** NSAIDs (with caution), weak opioids **only** if NSAID not tolerated / ineffective

→ **Treat underlying problems:** sleep disturbance / depression

→ **If chronic:** Ix to find underlying cause & Tx appropriately

- X-ray/CT/MRI
- Bone scan
- FBC, ESR/CRP, biochemistry

**TUMOURS:** → red flag symptoms & bony pain over a single spinal segment

**Primary:** meningioma, astrocytoma

**Secondary:** breast, lung, renal, prostate, bowel, thyroid

**SYRINGOMYELIA:** → fluid-filled cyst (syrinx) within spinal cord

**Causes:** Chiari malformation, spinal cord tumour, spinal cord injury, spinal cord infection e.g. meningitis

**Symptoms:**

- Pain & stiffness in neck, shoulders, arms & back
- Muscle wasting & weakness
- Loss of sensation & reflexes
- Painless burning sensation in fingers
- Headaches

**Diagnosis:** MRI/CT

**Management:**

- Drain syrinx
- Correct underlying cause

---

[2] NICE (2016, updated 2020) *Low back pain and sciatica in over 16s* [NG59]

# Peripheral nerve entrapment

## Carpal tunnel syndrome (CTS)

**CAUSE:**

- ↑ pressure on **median nerve** as it passes through **carpal tunnel at wrist**
- Roof of carpal tunnel = **transverse carpal ligament / flexor retinaculum**
- May be due to tendon swelling

**SYMPTOMS:**

- **Tingling/pain/numbness** in **radial 4 digits (not pinkie)** → may radiate into forearm
- Worse at night → wakes them up (may be because sleep with wrist flexed)
- Provoked by gripping
- Relieved by shaking hand / dangling over side of bed / raising hand
- May have reduced coordination → clumsy / drop things

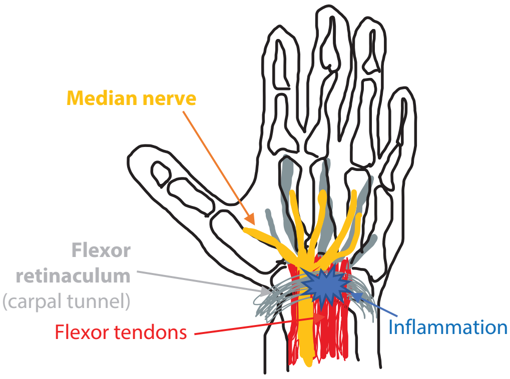

**Fig. 18.10** Carpal tunnel anatomy.

**INVESTIGATIONS: clinical diagnosis**

- Hx & examination (check for thenar wasting, motor & sensory function)
- **Nerve conduction studies** (if still uncertain → slowed conduction)

**MANAGEMENT[3]:** explain Dx & options for Mx

- Activity modification (avoid triggers, better wrist position)
- Splint wrist at night
- NSAIDs ± steroid injection
- Optimise underlying conditions e.g. thyroid disease / DM
- **Surgery:** carpal tunnel release if persistent symptoms >6w = high success rate
  - No surgery = likely wasting & numbness will worsen
  - Surgery should improve tingling/pain but may take 1y
  - Muscle weakness unlikely to improve but won't worsen

## Cubital tunnel syndrome

**CAUSE:** increased pressure on **ulnar nerve** at **elbow**

**SYMPTOMS:**

- Tingling/pain/numbness in 5th finger → may radiate into forearm
  → Exacerbated with prolonged elbow flexion e.g. using a phone
- ± Claw hand (only 4th & 5th fingers)

**MANAGEMENT:** similar to carpal tunnel syndrome

## Morton's neuroma

**CAUSE:** inflammatory lesion of **digital nerve** due to irritation/compression e.g. wearing tight shoes

**SYMPTOMS:**

- Pain / burning between toes / ball of foot
- Tingling / sensory loss between toes

**MANAGEMENT:**

- Offloading insoles
- Steroid injection
- Surgical incision of digital nerve

CTS = very common
(esp. middle-aged women)

**Risk factors for CTS:**

- pregnancy
- DM
- thyroid disease
- wrist fracture
- wrist arthritis
- **idiopathic**

***inflammatory arthritis***

5% of RA cases present with CTS prior to joint symptoms

**Contents of carpal tunnel:**

**9 tendons:** 4 FDS, 4 FDP, FPL
**1 nerve:** median nerve

NB: flexor carpi radialis has own sheath so is NOT part of carpal tunnel

**Special tests for carpal tunnel:**

→ Tinel test (at wrist)
→ Phalen's test
→ Durkan's test
→ Resisted thumb abduction

**Special tests for cubital tunnel:**

→ Tinel test (at elbow)
→ Pitres–Testut test
→ Froment's sign

**Guyon's canal syndrome**

Compression of the **distal ulnar nerve** at 'Guyon's canal', where ulnar nerve passes through a fibro-osseous tunnel at the **medial wrist**

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[3] RCOS Commissioning Guide (2017) *Treatment of carpal tunnel syndrome*

# Fibromyalgia

→ diagnosis made on specific criteria, characterised by **chronic widespread MSK pain & fatigue**

## Pathophysiology

Lack of sleep causes ↑ sensitivity to pain (any cause of ↓ sleep can trigger the condition)

## Clinical presentation

mainly women 40–60y

- **Widespread pain** ('pain all over')
- **Fatigue**
- **Unrefreshing sleep** – <6h sleep, early waking, vivid dreams

### ASSOCIATED FUNCTIONAL ILLNESSES:

- **IBS:** abdo pain, nausea, constipation/diarrhoea
- **Chronic migraine**
- **Chronic pelvic pain**
- **Chronic fatigue syndrome:** top DDx → MSK pain is less prominent

### OTHER (SOMATIC) SYMPTOMS:

- Depression, anxiety
- Dizziness, numbness, tingling
- Cold sensitivity / Raynaud's
- Dry eyes, sun sensitivity, rash
- Altered taste, sensitivity to smells
- Noise sensitivity

## Investigations

→ Screen for comorbidities e.g. depression

→ Find / rule out underlying causes e.g. RA

try to minimise Ix

## Management[4]

### EDUCATION:

→ **Reassure** → no serious underlying pathology

→ Nature of the condition → relapsing & remitting, no easy cure

→ 'Pacing' → have 'good' & 'bad' days → **do not** over-exert on good days (makes bad days worse!)

→ Sleep hygiene

**EXERCISE:** long-term graded exercise programmes

**CBT:** for pain-related depression / anxiety / catastrophising

### PHARMACOLOGICAL:

→ **Sleep:** low dose TCAs

→ **Pain:** paracetamol, low dose TCAs, pregabalin, duloxetine, tramadol

**Symptom severity score (SSS):**
ranks severity of associated Sx out of 3

1. Fatigue
2. Unrefreshing sleep
3. Cognitive Sx
4. No. of somatic Sx

(gives a score out of 12)

**Widespread pain index (WPI):**
19 areas in which patient has had pain over the past week e.g. left hip
(gives a score out of 19)

**Diagnostic criteria:** (all other Ix are normal)

1. Widespread pain involving **all 4 quadrants** of the body
2. Scoring **≥7 on the WPI** and **≥5 on SSS** or **3–6 on the WPI** and **≥9 on SSS**
   (Previously ≥11/18 recognised tender/trigger points)
3. Present **for ≥3m**

**Differentials:**
Must consider and rule out, as fibromyalgia often comorbid with serious rheumatological conditions

- Chronic fatigue syndrome
- SLE, MS, PMR
- RA, axial spondyloarthritis
- Hypothyroidism, T2DM, anaemia

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[4] Macfarlane GR, *et al.* (2017) EULAR revised recommendations for management of fibromyalgia. *Ann Rheum Dis*, 76:318

# Crystal-associated arthritis

## Gout

**AETIOLOGY** → pathological reaction of joint / periarticular tissues to **monosodium urate monohydrate crystals** (MSUM)
→ arises when there is prolonged hyperuricaemia

**Primary gout:** >90% due to inherited defect of uric acid excretion

- Almost exclusively males >40y

**Secondary gout:** chronic hyperuricaemia due to renal impairment or drug therapy

- Mainly >65y & post-menopausal women

**PRESENTATION:**

1. **Initially:** acute mono-arthritis in distal joint **(50% in MTPJ of big toe)**
   - Sudden pain, with red, hot, swollen joint
   - Often wakes patient in early morning
   - ± fever/malaise

   → Self-limiting over 5–14d (with itching & desquamation)
2. **Untreated:** recurrent attacks with reducing time between
   - Progressive cartilage & bone erosion (deformities)
   - Inflammatory polyarthritis in feet/hands/wrists
   - Deposition of palpable masses of urate crystals = **tophi**

**Chronic tophaceous gout:** usually takes 10y
**Deposits around** finger, hands, forearm, elbows, Achilles tendon, helix of the ear

**Differentials of gout:**

- Septic arthritis
- RA
- Other crystal arthropathies

**Sites affected by gout:** smaller joints
toe → ankle → midfoot → knee → hands → wrist → elbow

**Risk factors:**

| Primary gout | Secondary gout |
|---|---|
| • Male<br>• Age | |
| • FHx<br>• Metabolic syndrome<br>(↑ TGs, obesity, HTN, DM)<br>• High alcohol intake<br>(especially beer)<br>• High protein diet<br>(meat, seafood) | **Reduced excretion:**<br>• CKD<br>• Thiazide diuretics<br>• NSAIDs/aspirin<br>• Cytotoxic drugs<br>**Increased production:**<br>• Myeloproliferative disorders |

## Pseudogout

**AETIOLOGY** → **calcium pyrophosphate** crystal deposition (CPPD)

- **Idiopathic/sporadic:** most common
- **Secondary:** OA, trauma, dehydration, inherited metabolic disorder (e.g. haemochromatosis, hypomagnesaemia)

**PRESENTATION:**

1. **Usually CPPD is asymptomatic:** incidental finding of **chondrocalcinosis** on radiograph
2. **Acute attack (pseudogout):** similar symptoms to gout **(most cases in knee)**

**Sites affected by CPPD:** larger joints
Knee → wrist → elbows → pelvis/hips → shoulders

## Calcific periarthritis

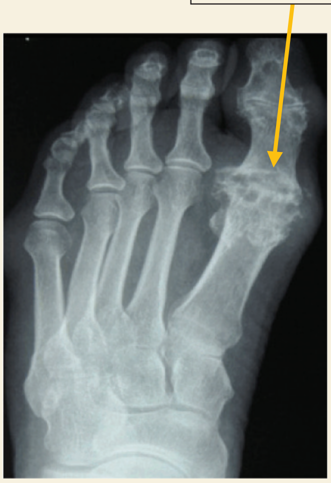
**AETIOLOGY** → **hydroxyapatite deposition** in periarticular tissues (muscles, cartilage etc.)

**PRESENTATION:**

1. **Acute attack:** spontaneously or following trauma
   - Extreme pain & tenderness, swelling & erythema

   → spontaneous resolution in 1–3w

Most common site: supraspinatus tendon



**Gout:** gouty erosions + tophi

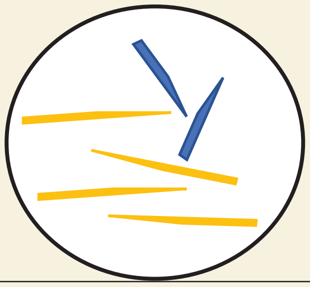
**Fig. 18.11** Radiograph showing gout in MTPJ of big toe.

## DIAGNOSIS OF CRYSTAL ARTHROPATHIES:

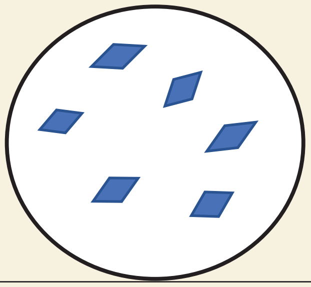
1. **Joint aspiration for polarised light microscopy:** confirms Dx
    - MSUM crystals present in gout, CPPD in pseudogout
    - Increased turbidity of synovial fluid (due to ↑WCC)
    - Often blood-stained/rust-coloured in **pseudogout**
2. **Serum urate levels:** hyperuricaemia is not diagnostic
    - Levels may be low in attack as crystals have deposited in joints
3. **Further assessment:** determine cause
    - FBC, ESR, CRP (↑WCC & CRP in acute attack)
    - Renal function (serum Cr, urine dip)
    - BP
    - Blood glucose
    - Serum lipid profile
4. **Consider X-ray:** for erosions/tophi

## MANAGEMENT OF CRYSTAL ARTHROPATHIES[5]:

1. **Acute attack:** do not start allopurinol for gout until acute attack has resolved
    - Pain relief: fast-acting oral NSAID (or oral colchicine if NSAID contraindicated) Causes N&V + diarrhoea
    - Stop attack: joint aspiration & intra-articular steroid injection
2. **Long-term treatment (for gout only):** aim to lower serum urate <360micromol/L
    - **1st line:** allopurinol OD (**xanthine oxidase inhibitor** → reduced urate production)
    - **2nd line:** febuxostat (avoid in cardiovascular disease)
    - Monitor serum urate monthly
3. **Lifestyle factors:**
    - Reduce alcohol & eat healthily & ensure drinking plenty of fluids
    - Weight management
    - Optimise control of comorbidities (DM, HTN)
    - Medication review (consider alternative diuretic / stop NSAIDs)



**Gout (MSUM) crystals under microscopy:**
Needle-shaped, **strong negative birefringence** yellow when parallel to ray



**Pseudogout (CPPD) crystals under microscopy:**
Rhomboid, **weak positive birefringence** **blue** when parallel to ray

**Side-effects of allopurinol:**

- Skin rash, N&V
- Headaches, drowsiness
- Altered taste

**Avoid with azathioprine**

NB: allopurinol can initially trigger attacks due to dissolution of crystals

**Indications for long-term Tx:**

- Recurrent attacks
- Evidence of bone/joint damage
- Tophi
- Associated renal disease
- Severely elevated serum urate

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[5] NICE (2022) *Gout: diagnosis and management* [NG219]

# Rheumatoid arthritis

Affects 1% of the population – F>M

→ Common systemic inflammatory disease affecting **synovial joint lining** to cause **symmetrical polyarthritis**

→ **Autoimmune destruction** of cartilage & bone

→ **Risk factors:** age (40s), female, FHx, **smoking**, obesity

## Presentation

- Hot, swollen joint
- Joint pain
- Stiffness – worse in mornings **(lasts >1h)**
- Loss of function of joint

→ **Systemic:** malaise, fatigue, fever, weight loss

## Extra-articular features

1. **Lungs:** fibrosis, pleural effusions
2. **CVS:** pericarditis, atherosclerosis (↑ **risk MI/stroke)**
3. **Eyes:** episcleritis, scleritis, keratoconjunctivitis
4. **Vasculitis:** splinter haemorrhages (rare), vasculitic ulcers
5. **Peripheral neuropathy**
6. **Atlanto-axial subluxation**
7. **Other:** Sjögren's syndrome, Felty's syndrome* *RA + enlarged spleen + neutropenia

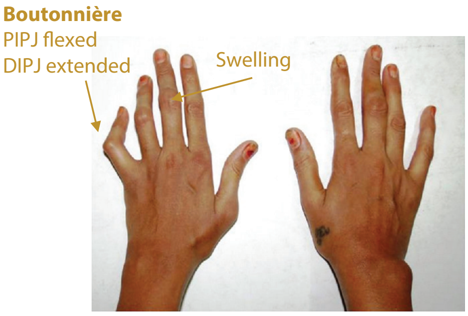

**Fig. 18.12** Hand deformities.

## Investigations

→ thorough Hx & examination

- **FBC:** ↑ CRP, ESR, platelets (inflammation) (↑ WCC suggests infection) ↓ Hb = normocytic, normochromic anaemia
- **Rheumatoid factor (RF):** not specific
- **ANA antibody**
- **Anti-CCP antibodies: specific**
- **X-ray joints:** soft tissue swelling, periarticular erosions

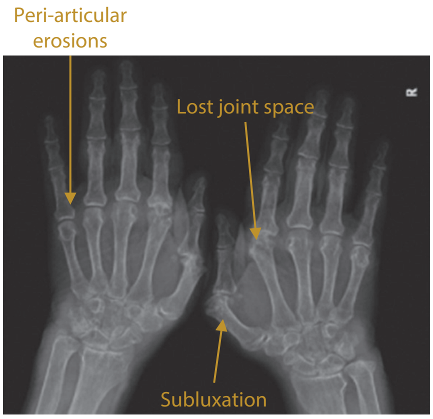

**Fig. 18.13** Radiograph showing features of RA hands.

**To assess suitability for Tx:**
Kidney & liver function + CXR

**To rule out other conditions:**

- **Joint aspirate:** r/o infection & crystal arthropathies
- **Serum uric acid:** r/o crystal arthropathies

**Differential diagnosis:**

- Rheumatoid arthritis
- Septic arthritis
- Crystal arthropathies (pseudo/gout)
- CTDs (SLE, vasculitis)
- Spondyloarthropathies ('PEAR') →

**P**soriatic
**E**nteropathic
**A**nkylosing spondylitis
**R**eactive

**Joint involvement:** usually **symmetrical**

- **Small joints of hands & feet** (MCPJ, PIPJ, MTPJ)* *DIPJs spared
- Wrists & ankles

→ Can affect any other synovial joints as well
→ Spine usually spared

**Atlanto-axial subluxation:**

→ Synovitis of cervical spine causing ligament damage & instability
= subluxation of atlanto-axial joint (C1 & C2)

**Sx:** neck stiffness/pain, neuro Sx (weak/numb/tingling)
**Dx:** MRI

**Antibody sensitivity & specificity**

| | RF | Anti-CCP |
|---|---|---|
| **Sensitivity** | 60–80% | 60–65% |
| **Specificity** | 60–70% | >90% |

**Other causes of RF +ve**

- EBV, parvovirus, TB
- Hepatitis B, syphilis
- Sarcoidosis
- SLE, Sjögren's
- Dermatomyositis

Management involves an MDT approach
→ emphasis on early Dx, referral & Tx
→ treatment is lifelong

**Assessing severity:** guide & monitor Tx

**DAS28 score:**

- Joints involved ( /28)
- CRP/ESR or no. swollen joints
- Global health

**Mod:** >3.2 **High:** >5.1

**Biologics indicated if:**
→ DAS28 >5.1 **and**
→ Trialled ≥2 DMARDs (one being MTX)

**Ix before starting Tx:**

- FBC, LFT, renal function
- CRP/ESR
- CXR for pulmonary fibrosis
- HIV/hepatitis screen if high risk
- TB screen – biologics can reactivate

**Role of primary care:**

- Annual review of Sx, comorbidities, Tx
- Blood monitoring
- Information & education
- Managing flares

## Management[6]

### 1. PHARMACOLOGICAL

*contraindicated in PUD, CVD, renal impairment

- **Analgesics**
  → NSAIDs* (ibuprofen, aspirin, naproxen etc.)
  → Glucocorticoid injections (IM/IA): (prednisolone) = while waiting for DMARDs to work / during flares
- **DMARDs (disease-modifying antirheumatic drugs)**
  **1st-line = PO/IM methotrexate once WEEKLY (+ folate)**
  → Interfere with immune response to **slow disease progression**
  → Take 6–12w to become clinically effective
  → **Regular blood monitoring:** FBC, U&Es & LFTs (fortnightly for 6w, then monthly)
  → Counsel on SEs / red flags: N&V, sore throat, mouth ulcers, infections
  → Can add additional DMARD if disease activity remains high e.g. hydroxychloroquine, sulfasalazine
- **Biologics:** anti-TNF-α (e.g. IV infliximab, IL-6 inhibitors, SC etanercept)

NB: if untreated, long-term inflammation can cause OA

**Things to consider when selecting DMARDs / biologic therapy:**

- Pregnancy / family planning – sulfasalazine & hydroxychloroquine are safe to use
- Alcohol consumption – leflunomide and methotrexate contraindicated
- Pulmonary fibrosis – methotrexate, leflunomide and anti-TNF contraindicated
- History of cancer (site, type, years in remission) – biologics contraindicated
- Renal/hepatic health – important for all medications
- General health / comorbidities – important for all medications

Caution with sulfasalazine if allergy to aspirin, co-trimoxazole, sulphonamides

### 2. NON-PHARMACOLOGICAL

- **Physiotherapy:** education, exercise/hydrotherapy, pain relief (heat packs, acupuncture)
- **Occupational therapy:** adaptive skills, equipment, splints, joint protection, activity modification
- **Patient education:** adapt activities / conserve energy, sleep hygiene, exercise, smoking cessation, weight loss

Patients have ↑CVS risk & important to counsel on this

### 3. SURGERY

Consider referral for surgical review if:

- Persistent pain due to joint damage
- Worsening joint function
- Progressive deformity

**NB: Surgery needs careful negotiation with patient:** warn that improvements in function post-surgery may be minimal, and temporarily withholding treatment may precipitate a flare in their condition

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[6] NICE (2018, updated 2020) *Rheumatoid arthritis in adults* [NG100]

# Seronegative spondyloarthritis

## Shared clinical features

- **Asymmetrical mono-/oligoarthritis** of large joints (including **spine & SIJ**)
- **Affects tendons (enthesitis):** plantar fasciitis, Achilles tendinitis
- **Extra-articular Sx:** iritis, palmar/plantar rashes, dactylitis, mouth ulcers, IBD

**Investigations to r/o other arthropathies:**

1. FBC, CRP, ESR (not always raised)
2. RF & ANA/CCP antibodies (r/o RA)
3. Joint aspirate (r/o infection & gout)

## Psoriatic arthritis

**Clinical presentation:** many different patterns of *pain, stiffness, swelling*

- **Asymmetrical** oligoarthritis of **weight-bearing joints (sacroiliac) & DIPJs** (mimics OA)
- **or symmetrical** polyarthritis (mimics RA)
- **or spondylitis** (mimics ankylosing spondylitis)
- **or arthritis mutilans** (dactylitis & telescoping of digits)

Presents at 35–55y

**Diagnosis:**

- Clinical features (e.g. dactylitis)
- Lack of RF/ANA/CPP
- X-ray (bony spurs)

DAS 66/68 assesses severity

**Management:** similar to RA (NSAIDs, DMARDs (if peripheral joints), biologics) **or** if features of ankylosing spondylitis (SIJ/spinal involvement) treat as below

## Ankylosis spondylitis

**Clinical presentation:**

Presents at 20–30y, M>F

- Insidious **lower back & buttock pain** (better with exercise)
- Morning stiffness & restricted spinal motion
- ± extra-articular features

→ **Later:** loss of lumbar lordosis & ↑ thoracic kyphosis

**Examination:**

- Examine for sacroiliac tenderness
- Assess spinal movements, chest expansion & lordosis

→ modified Schober's (mark points on back & flex → increase of 5–7cm = normal)

**Diagnosis:**

**MRI = gold standard** (+ X-ray/CT)

- Inflammatory lesions in corners of vertebral bodies
- **Syndesmophytes** (bony bridges) between vertebrae
- Sclerosis of sacroiliac joints (up to 10y after symptom onset)

X-ray changes don't appear for 5–7y

**Bloods:**

- May have mildly raised ESR but normal CRP (inflammation not infection)

**Management[7]:**

1. **Physio & OT:** regular exercises, deep breathing, activity modification
2. **Analgesia:** strong NSAIDs
3. **Anti-TNF-α:** after tried 2 NSAIDs and BASDAI score still high
4. **Surgery:** only if severe/progressing despite optimal non-surgical treatment **and** severely affecting quality of life*

*BASDAI & BASFI scores: assess mental health & functional ability

→ A group of **inflammatory arthropathies**
→ **no serum markers** but association with **HLA-B27** (FHx)

**Types:** 'PEAR'
- **P**soriatic
- **E**nteropathic (IBD)
- **A**nkylosing spondylitis
- **R**eactive

**Extra-articular features of PsA:**
- Psoriatic rash (**not always present**)
- Nail changes (pitting, onycholysis)

**Extra-articular features of AkS:**
- Anterior uveitis (30%)
- IBD
- Apical pulmonary fibrosis
- Restrictive lung disease (↓ expansion)
- Cauda equina syndrome (rare)

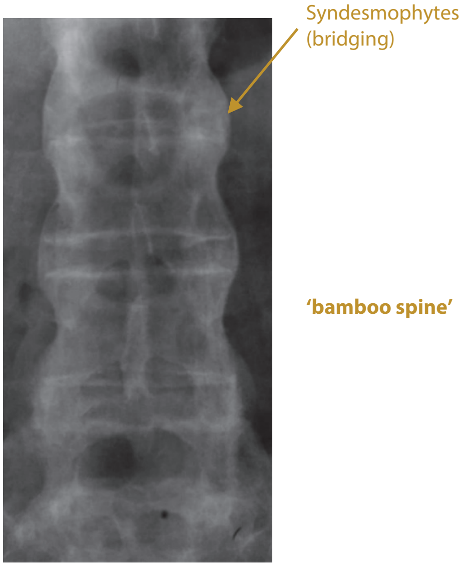

**Fig. 18.14** Radiograph of spine in AkS.

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[7] NICE (2017) *Spondyloarthritis in over 16s: diagnosis and management* [NG65]

## Reactive arthritis

→ Triggered by **enteric or genitourinary (STIs) infection**

**Clinical presentation:**

- Acute asymmetrical oligoarthritis of **lower limbs / back** → backache
- **Hot, swollen joints** (DDx: septic arthritis & pseudo/gout)
- **Fever & malaise**
- ± extra-articular features

**Investigations:**

→ Diagnosis mainly on **Hx of preceding infection (2–4w earlier)**

- CRP, ESR = largely elevated
- WCC = elevated if infection still present
- Stool & urine cultures + genitourinary swab = for cause
- IgM/IgA/IgG = raised/rising
- **Joint aspiration** – r/o septic arthritis & pseudo/gout

**Management:**

- Full dose NSAIDs (analgesia)
- Intra-articular steroid injections
- ABX **if identified organism**

**Causative organisms:**

- Salmonella, *E. coli*, campylobacter, shigella
- *Chlamydia trachomatis*, gonorrhoea
- Streptococcus

**Extra-articular features:**

- **Reiter's triad:** conjunctivitis, urethritis, arthritis 'can't see, can't wee, can't bend the knee'
- Skin rashes / erythema nodosum

## Enteropathic arthritis

→ **in 10–15% of those with IBD**

**Clinical presentation:**

- Joint swelling, pain, stiffness
  - **Type I:** oligoarticular & asymmetrical + active IBD
  - **Type II:** polyarticular & symmetrical
- ± extra-articular features

**Diagnosis:** confirm presence of IBD with X-ray & endoscopy + biopsy

**Management:**

1. **Treat IBD**
2. **Treat arthritis**
   - Use NSAIDs with caution as can worsen IBD
   - DMARDs: sulfasalazine is 1st line as helps IBD
   - Anti-TNF-α

**Extra-articular features:**

- **IBD features**
- Iritis & skin rashes / erythema nodosum

# Multisystem connective tissue disease

→ autoimmune conditions (associate with **ANA antibody**)
→ can present with a huge range of symptoms e.g. myalgia, fatigue, rashes, photosensitivity, mouth ulcers, indigestion

**Characteristic pathological features (4):**

1. Inflammation (widespread aches/pains)
2. Fibrosis (skin lesions)
3. Vasospasm (often get 2° Raynaud's)
4. Thrombosis (nail infarcts, VTE)

| | SLE | Dm/Pm | Sjögren's | Scleroderma | APS |
|---|---|---|---|---|---|
| **Inflammation** | +++ | +++ | ++ | +/– | – |
| **Fibrosis** | + | ++ | ++ | +++ | – |
| **Vasospasm** | ++ | + | + | +++ | – |
| **Thrombosis** | ++ | +/– | +/– | + | +++ |

## Systemic lupus erythematosus (SLE)

**Clinical presentation:** relapsing & remitting

- **Skin:**
  - **Malar rash** (photosensitive)
  - Discoid rash
  - Mucosal ulcers
- **Vascular:** nail-edge infarcts, splinter haemorrhages, Raynaud's
- **MSK: symmetrical polyarthritis** (similar to RA)
- **Lungs:** pleuritis (SOB)
- **Heart:** pericarditis (chest pain)
- **CNS:** seizures, psychosis, anxiety/depression
- **Renal:** nephritis (proteinuria & cast cells in urine)
- **Haematological:** pancytopenia, lymphadenopathy, organomegaly

+ Non-specific systemic upset: malaise, low fever, weight loss, fatigue

**Investigations:** history = key to Dx

- BP & urinalysis – possible haematuria/proteinuria
- FBC – ↓WCC, ↓platelets, haemolytic anaemia
- CRP, ESR (**CRP may be normal while ESR raised**)
- U&Es, LFTs – renal biopsy if abnormal
- Complement levels – decreased
- **ANA, RNP** (not specific for SLE)
- **dsDNA, anti-SM** (specific for SLE)
- **anti-Ro & anti-La** (specific for SLE & Sjögren's)
- Antiphospholipid Abs & lupus anticoagulant test
- CXR

Anti-Ro/La often seen in ANA-negative SLE

**Management:**

→ **Skin:** topical steroids ± hydroxychloroquine + vitamin D
→ **Joints (mild):** NSAIDs, hydroxychloroquine
→ **Joints (mod/severe):** low dose steroids (PO/IM)
→ **Severe flares:** strong immunosuppression (DMARDs e.g. cyclophosphamide)

+ **Lifestyle advice:** high SPF sun cream, regular exercise, avoid UV
+ **Monitoring:** bone health, urinalysis, BP

**Red flags of CTDs:**

- BP >230/120
- casts in urine
- SOB
- CNS signs
- severe abdo pain
- ischaemic vasculitis

**Epidemiology of SLE:**

- **Peak age:** 35–45y & 60s
- 1 in 4000 people
- F:M = 10:1

**Risk factors for SLE:**

- Female
- Afro-Caribbean/Asian
- FHx
- UV light
- Oestrogens
- Stress

Trigger relapses

**Complications of SLE:**

- Thromboembolic disease
- Atherosclerosis
- Infection

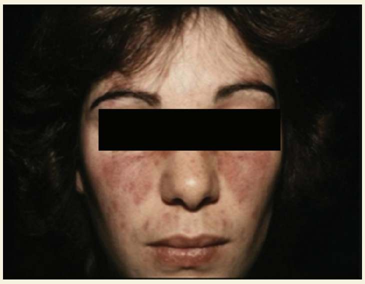
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**Fig. 18.15** Malar rash.

## Antiphospholipid syndrome (APS)

→ *tendency to* ***recurrent thrombosis*** *&/or persistently ↑ antiphospholipid Abs*

**Clinical presentation:** 'CLOT'
**C**oagulation defect (arterial or venous thrombosis)
**L**ivedo reticularis (± chronic leg ulcers)
**O**bstetric issues (recurrent miscarriage)
**T**hrombocytopenia (in 1/3 cases)

20–30% cases associated with SLE

**Investigations:** r/o other clotting disorders

- FBC & coagulation screen
- Antiphospholipid Ab & lupus anticoagulant test

**Management[8]:**
→ **Antiphospholipid Ab +ve, but no clinical thrombosis:**
Low dose aspirin & lifestyle advice (stop smoking, avoid COCP)
→ **Recurrent miscarriage:** low dose aspirin + LMWH
→ **Thrombotic event:** lifelong warfarin

## Scleroderma (systemic sclerosis)

→ ***vascular damage & fibrosis*** *within the skin & organs due to excess collagen*

**Clinical presentation:**

- Skin fibrosis – thickening, tightness, hyper-/hypopigmentation, joint contractures, fingertip fissures
- Other organ involvement – fibrosis of GI tract, heart, lungs, kidneys

### LIMITED SCLERODERMA:

→ peripheral skin involvement (distal to the elbows & knees)
→ anti-centromere antibodies

**Clinical presentation:** 'CREST'
**Calcinosis:** calcium deposits under skin
**Raynaud's**
**Esophageal dysmotility:** heartburn
**Sclerodactyly:** thick, swollen fingers
**Telangiectasia:** on face/fingers

**Management:** no cure so Mx is supportive

- Skin: topical steroids
- HTN, pulmonary HTN & Raynaud's: vasodilators e.g. CCBs
- Reflux: PPIs
- Pain: ibuprofen, paracetamol

### DIFFUSE SCLERODERMA:

→ widespread skin changes (may involve whole body)
→ anti-Scl-70 antibodies

**Clinical presentation:** more severe organ fibrosis

- heart (arrhythmia/HF)
- lungs* (SOB, pulmonary HTN)
- kidneys (AKI & HTN)

**Mx of pulmonary fibrosis: immunosuppression (IV cyclophosphamide), transplant*

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[8] Keeling D, *et al.* (2012) Guidelines on the investigation and management of antiphospholipid syndrome (2012) *Br J Haematol,* 157:47

## Sjögren's syndrome

→ ***autoimmune destruction & fibrosis** of exocrine glands*

**Clinical presentation:**

- **Dry eyes, mouth, vagina, skin**
- Dry throat/sinuses – cough
- Parotid swelling

**Extra-glandular features:**

- Polyarthritis/arthralgia
- Raynaud's / skin rashes
- Vasculitis
- Nephritis
- Peripheral neuropathy
- Increased risk of lymphoma

**Investigations:**

1. **Schirmer's test** – blotting paper inside lower eyelid to measure amount of moisture produced in 5min
2. **Antibodies:** anti-Ro, anti-La, ANA (r/o RA)
3. **FBC** (↓ WCC, anaemia) **& ESR** (often ↑)

**Management:**

- Artificial tears & saliva (± muscarinic agonists – pilocarpine)
- Frequent drinks, sugar-free pastilles

**Mx of extra-glandular features:**

- NSAIDs/hydroxychloroquine for arthralgia
- Immunosuppressants (steroids/cytotoxics)

**Autoantibodies present in Sjögren's:**
**anti-Ro** (anti-SSA)
**anti-La** (anti-SSB)

Good prognosis: slowly progressive

## Idiopathic inflammatory myopathies

→ *group of conditions causing **inflammation of striated muscle***

Polymyositis, dermatomyositis, inclusion body myositis

→ can occur alone or with other CTDs
→ they are very rare

**Clinical presentation:**

→ **Proximal muscle weakness** 3–6m onset

- Difficulty rising from chair, climbing stairs
- Difficulty brushing hair, getting dressed
- Waddling gait
- Dyspnoea/dysphagia (if pulmonary/GI muscles)

→ **Characteristic rash in dermatomyositis**

- Purple heliotrope rash – around eyelids
- Gottron's papules – scales over knuckles

All are associated with ↑ risk of malignancy

**Investigations:**

1. **FBC** (anaemia & ↑ ESR)
2. **CK** = elevated in most
3. **ANA, RF & MSAs** = often positive
4. **Electromyograph** – spontaneous muscle fibrillation
5. **Muscle biopsy** = diagnostic
6. **MRI** – detect muscle inflammation

muscle fibre necrosis + inflammation infiltrate

**Management:**

- **High dose corticosteroids** (1m prednisolone)
- Other immunosuppressants:

+ exercise rehabilitation, physiotherapy & occupational therapy

**Inclusion body myositis (IBM):**

Affects males >50y (rare)

- **Asymmetrical** progressive muscle weakness (often quads)
- **Inclusion body** (protein clumps) on biopsy

**Autoantibodies present in IIM:**
**ANA & RF**
**MSAs** (myositis-specific Abs)

# Systemic vasculitis

**Inflammation of vessel walls → may cause aneurysm/rupture or occlusion & organ damage**
→ Can be **primary or secondary** to underlying disease/drug-induced
→ Classified based on **size of smallest vessel** involved (small, medium, large)

**Examples of underlying disease:**
RA, CTD, infection (Hep B/C), haematological malignancy

## Clinical features

| System | Symptoms |
|---|---|
| **Systemic** | Malaise, myalgia, arthralgia/arthritis, headache, fever, weight loss |
| **Cutaneous** | Purpura, infarct, ulcer, gangrene |
| **Mucous membs** | Ulcers, conjunctivitis/scleritis/episcleritis/uveitis, retinal haemorrhage |
| **ENT** | Epistaxis, nasal crusting, stridor, hearing loss |
| **Respiratory** | Dyspnoea, haemoptysis |
| **Cardiovascular** | Pericarditis, angina/MI, thrombosis, bruits |
| **Gastrointestinal** | Abdo pain, bloody diarrhoea, perforation |
| **Renal** | HTN, proteinuria, haematuria, casts, renal failure |
| **Neurological** | Stroke, seizures, confusion |

pattern of Sx depends on specific type of vasculitis

**Autoantibodies are often present:**
**ANCA (PR3 OR MPO)**
(+ raised complement)

## Investigations

→ **Thorough Hx & examination**
- FBC, U&Es, LFTs = raised creatinine if renal failure
- Urinalysis = may be protein, blood, casts
- **ESR/CRP & complement levels = often raised**
- **ANCA = often present**
- Organ biopsies = may be diagnostic
- Angiography = may show stenosis/aneurysms

## Management

→ **Large vessel:** steroids usually enough
→ **Medium/small vessel:** IV cyclophosphamide + steroids (more intense immunosuppression)

## Types of vasculitis

| | Vasculitis | Epidemiology | Symptoms | Management |
|---|---|---|---|---|
| Large vessel | **GCA** | Elderly (>50y)<br>Caucasians | Headache, scalp tenderness, jaw claudication, blurred/painful vision, ↑ESR | **Emergency:** optic nerve ischaemia<br>High dose corticosteroids ≥1y |
| | **Takayasu's arteritis** | Females <50y<br>Asians | **Loss of pulses**, bruits, aortic occlusion, intermittent claudication | Corticosteroids |
| Medium vessel | **Kawasaki disease** | Children <5y | Fever, red/swollen mucous membranes, skin desquamation | High dose aspirin + IV immunoglobulins |
| | **Polyarteritis nodosa** | Adults | Multisystem (but **not kidneys**), aneurysms,<br>often Hep B +ve + '**beads on a string**' angiogram | Corticosteroids + antivirals |
| Small vessel | **Granulomatosis with polyangiitis*** | Mainly adults | **Upper resp** – epistaxis, nasal crusting **(saddle nose)**<br>**Lower resp** – dyspnoea, haemoptysis<br>**Kidney** – glomerulonephritis<br>Predominantly cANCA/**PR3-ANCA** | Corticosteroids + cyclophosphamide |
| | **Churg–Strauss syndrome** (eosinophilic) | Mainly adults | **Respiratory tract** – asthma, allergic rhinitis<br>Multiple drug allergies<br>Predominantly pANCA/**MPO-ANCA** eosinophilia | Corticosteroids + cyclophosphamide |
| | **HSP** | Mainly children | Purpura, arthritis, abdo pain, haematuria<br>IgA complexes | Self-limiting |

*formerly known as Wegener's granulomatosis

# Polymyalgia rheumatica

→ mild periarticular inflammation
→ common (3% of >65s, and almost never <50y), F:M = 3:1

## Clinical features

**Fairly acute onset of:**

- **Bilateral** stiffness & aching of **proximal joints** (shoulders, hips)
  → struggle to climb stairs / reach above head / turn in bed / sit on toilet
  → limited due to pain/stiffness, NOT MUSCLE WEAKNESS
- Morning stiffness >45min (eases with use)
- Evidence of acute phase response (**↑ ESR, fatigue, fever, night sweats, weight loss)**

± **symptoms of GCA** (headache, jaw pain etc.) → **always ask for these in history** as GCA needs urgent Tx

**Often a big psychological impact** – loss of independence / active life → fear of more serious / terminal illness

## Investigations

**PMR = diagnosis of exclusion** → tests depend on how unwell patient is

**Initial investigations:**

- FBC, CRP/ESR – may have anaemia, **↑ESR/CRP & platelets**
- U&Es, LFTs, TFTs
- **Bone profile**: r/o **↓ vit D** as cause of myalgia
- RF, ANA, anti-CCP = **negative**
- Creatine kinase = **normal** (r/o inflammatory myopathies)

**Other investigations to consider:**

- Muscle biopsy = **normal**
- USS – may show bursitis/synovitis
- Joint X-ray – r/o RA, OA etc.
- CXR – r/o malignancy, TB
- MRI/CT – r/o malignancy
  → Assess for GCA

↑ Ca may suggest malignancy

## Management[9]

1. **Low dose prednisolone** (10–20mg for 2w then wean down slowly to maintenance dose for 1–2y)
   → usually **improve within 48h** & inflammatory markers normalise within 1m
2. **Co-prescribe bone protection** (bisphosphonate) **& PPI** with long-term steroids
3. **Follow-up appointments:**
   - Wean down steroid dose
   - Assess disease activity & response to Tx
   - Monitor for Tx side-effects (BP, glucose, bone health, eye health etc.)

**Strong link with GCA**
15–30% of those with PMR have GCA
50% of those with GCA have PMR

*Patients will often describe the functional impact on ADLs rather than specific Sx*

*"I feel like I've aged 20 years!"*

**Differentials: need to r/o serious DDx**

- RA/OA
- CTDs e.g. SLE
- Crystal arthropathies
- Inflammatory myopathies
- Cervical spondylosis
- Fibromyalgia
- Thyroid/parathyroid disease
- Low vitamin D
- Infection/sepsis
- Multiple myeloma
- Malignancies/leukaemia

Can cause muscle weakness & pain

Presents with similar systemic symptoms + muscle pain/ache

**Refer to specialist if red flags:**

<60y, normal inflammatory markers, Tx-resistant, suspect malignancy / other rheumatological condition

**GIANT CELL ARTERITIS (GCA)** = large vessel vasculitis → **50% associate with polymyalgia rheumatica**

**Clinical presentation:** usually in those >60y

- Headache
- Scalp/temporal tenderness
- Jaw claudication
- Amaurosis fugax / sudden unilateral visual loss

± **PMR signs:** morning aching

± lethargy, depression, low grade fever, anorexia, night sweats

**Investigations:** r/o stroke & trauma

1. **Hx & exam** – vision, tenderness, PMR Sx
2. **ESR & CRP = raised** (also ↑ plts & ALP)
3. **Ix for cause / r/o stroke:** BP, glucose, cholesterol/TGs
4. **Temporal artery biopsy:** skip lesions so may be negative

**Management[10]:**

**High dose prednisolone (40–60mg/d PO)**
or IV methylprednisolone if rapidly progressing visual symptoms
→ Continue for around 2y, tapering down the dose

**Muscle pain + new onset headache, think PMR & GCA!**

→ Same day ophthalmology referral if eye symptoms
→ **Risk irreversible vision loss if untreated**

---

[9] NHSL (2019) *Guidelines for the management of polymyalgia rheumatica*
[10] NICE CKS (2022) *Clinical scenario – Management of giant cell arteritis*

# Miscellaneous musculoskeletal conditions

## The unstable shoulder

*→ abnormal movement of humeral head relative to glenoid*

| | **Stanmore classification[11]** | | |
|---|---|---|---|
| | **1. Traumatic structural**<br>e.g. contact sports | **2. Atraumatic structural**<br>e.g. repetitive injury / athletes | **3. Muscle patterning non-structural** |
| **History** | - Unilateral<br>- Hx of trauma/injury | - Bilateral or unilateral<br>- No single traumatic event | - Often **bilateral**<br>- No injury or repetitive movement |
| **Underlying pathology** | Damage to joint structures<br>= Bankart/Hill–Sachs lesions | Microtrauma to capsules/ligaments<br>= Bankart/Hill–Sachs lesions | Unbalanced muscle recruitment |

**Investigating instability:**

1. **History:** trauma, repetition etc.
2. **Examination:** apprehension tests, stability tests
3. **X-ray:** in 2 planes
4. **CT:** bony lesions
5. **MRI:** labrum, capsule, rotator cuff

### MANAGING INSTABILITY:

**Traumatic (type I):**

1. Sling immobilisation + avoiding risky activities for 2–3w
2. Physiotherapy
3. Surgical stabilisation/repair (if high risk recurrence)

**Atraumatic (type II & III):**

1. Physiotherapy & rehab (improve tone, muscle strength & proprioception)
2. Surgical repair (for type II if no improvement in 6m → **assess suitability with MRI first)**

Surgical intervention avoided in type III instability

## Hypermobility

*→ ligamentous laxity, with no evidence of rheumatological disease*

**Possible symptoms of hypermobility:**

**MSK:**
- Soft tissue **injury**
- Joint instability/subluxation
- Repetitive atraumatic dislocation
- Chronic, non-inflammatory joint pain
- Early OA development

**Extra-articular:**
- Stretch marks & easy bruising
- Hernias, prolapses, varicose veins
- Pain, fatigue, depression/anxiety
- PoTS (postural tachycardia syndrome)

**DIAGNOSIS: Beighton score[12] ≥ 4/9**

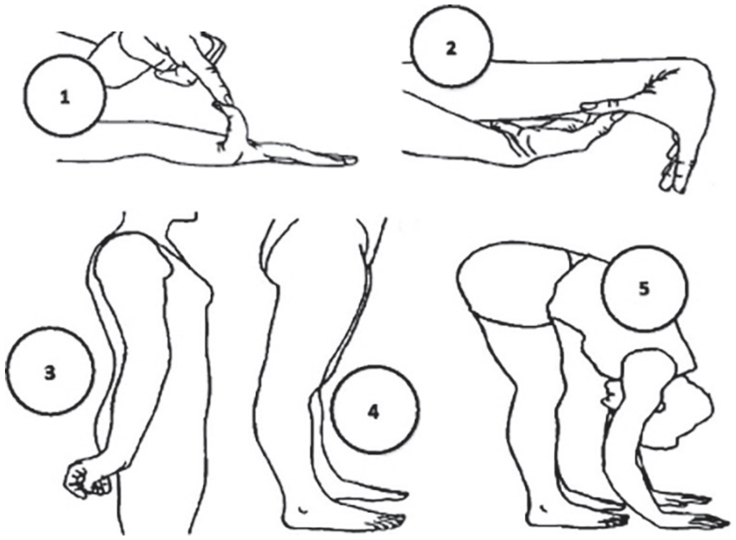

**Fig. 18.16** Movements assessed in the Beighton score.

---

[11] Jaggi A, Lambert S (2010) Rehabilitation for shoulder instability. *Br J Sports Med*, 44:333

[12] Grahame R, Bird HA, Child A (2000) The revised (Brighton 1998) criteria for the diagnosis of benign joint hypermobility syndrome (BJHS). *J Rheumatol*, 27:1777

## Neuropathic (Charcot) joint

*→ joint deformities secondary to peripheral neuropathies*

**PATHOGENESIS:**

→ osteoclast activation causing rapid bone destruction → deformities

**CLINICAL PRESENTATION:**

1. **Structural deformity** → commonly in feet → **high arched foot (pes cavus)** + hammer (clawed) toes
2. **Pain**
3. **Swelling, erythema, warmth**

**MANAGEMENT:** takes 18–24m to heal with rest

- Immobilisation of joint (halts inflammation)
- Custom shoes / bracing
- **Prevent ulceration & therefore ulcers**
- Surgery

**Causes of neuropathic joint:** 'SLASSH':

**S**ugar (diabetes)
**L**eprosy/syphilis
**A**lcohol
**S**yringomyelia
**S**pinal cord injury / spina bifida
**H**ereditary motor sensory neuropathy

# TRAUMA & ORTHOPAEDICS

# 19

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## ABBREVIATIONS

**#** – Fracture
**AC** – Acromioclavicular
**AKI** – Acute kidney injury
**ALP** – Alkaline phosphatase
**AP** – Anterior–posterior
**APS** – Antiphospholipid syndrome
**ARDS** – Acute respiratory distress syndrome
**ASIS** – Anterior superior iliac spine
**ATLS** – Advanced trauma life support
**AVN** – Avascular necrosis
**BMD** – Bone mineral density
**BAME*** – Black, Asian and minority ethnic
**CC** – Coracoclavicular
**CK** – Creatine kinase
**CMV** – Cytomegalovirus
**CRT** – Capillary refill time
**DCML** – Dorsal column medial lemniscus
**DDH** – Developmental dysplasia of the hip
**DEXA** – Dual-energy X-ray absorptiometry
**DHS** – Dynamic hip screw
**DIC** – Disseminated intravascular coagulation
**EBV** – Epstein–Barr virus
**EPL** – Extensor pollicis longus
**FAST** – Focused Assessment with Sonography in Trauma
**FFP** – Fresh frozen plasma
**FICB** – Fascia iliaca compartment block
**FNB** – Femoral nerve block
**FOOSH** – Fall on outstretched hand
**GCS** – Glasgow Coma Score/Scale
**ICP** – Intracranial pressure
**Ig** – Immunoglobulin
**IM nail** – Intra-medullary nail
**IVDU** – Intravenous drug user
**LOC** – Loss of consciousness
**LP** – Lumbar puncture
**LUQ** – Left upper quadrant
**MOI** – Mechanism of injury
**MRSA** – Methicillin-resistant *Staphylococcus aureus*
**NAI** – Non-accidental injury
**NOF** – Neck of femur
**OA** – Osteoarthritis
**OP** – Osteoporosis
**PA** – Posterior–anterior
**PPI** – Proton pump inhibitor
**PSA** – Prostate-specific antigen
**PTH** – Parathyroid hormone
**PVD** – Peripheral vascular disease
**RA** – Rheumatoid arthritis
**ROM** – Range of movement
**RTA** – Road traffic accident
**RUQ** – Right upper quadrant
**SA** – Surface area
**SAH** – Subarachnoid haemorrhage
**SCD** – Sickle cell disease
**SD** – Standard deviation
**SLE** – Systemic lupus erythematosus
**STI** – Sexually transmitted infection
**SUFE** – Slipped upper femoral epiphysis
**TXA** – Tranexamic acid
**UO** – Urine output
**URTI** – Upper respiratory tract infection

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*We appreciate that it is important to recognise each ethnic minority group separately, but for brevity and ease of use of this book we have used the abbreviation in a limited number of places.

# Bone and joint infection

**Most common 1° sources include:** URTI, UTI, STI & endocarditis

**Aetiology:** spread via blood (haematogenous), adjacent soft tissue infection or penetrating wound / procedure

NB: osteomyelitis can spread to cause septic arthritis

**Risk factors:**

Extremes of age, immunosuppressed, DM, recent ops/injections, IVDU, wounds, prostheses, RA, crystal arthropathy

| | **OSTEOMYELITIS** = infection of the **bones** | **SEPTIC ARTHRITIS (SA)** = infection of the **joint space** |
|---|---|---|
| **Organisms** | • ***Staph. aureus***<br>• *Pseudomonas*<br>• *Streptococcus*<br>• *Salmonella* (in SCD) | • ***Staph. aureus***<br>• ***N. gonorrhoeae*** (young adults)<br>• *Cutibacterium acnes* (shoulder)<br>• *Streptococcus*<br>• *E. coli* |
| **Location** | **Kids:** long bone metaphyses<br>**Elderly:** vertebrae | **Monoarticular** (often hip/knee) → **unlike in RA** |
| **Symptoms** | **Acute:**<br>• **Pain** (throbbing) & **immobility / loss of function**<br>• **Systemic Sx:** fever, rigors, malaise<br>• Swelling / sinus formation, erythema = later signs<br>→ **15% have coexistent septic arthritis**<br>**Chronic:**<br>• Periods of quiescence with exacerbations | • **Pain** (constant) & **immobility** (pseudoparesis)<br>• **Swelling, tender, warmth, erythema over joint**<br>• **Systemic Sx:** fever, rigors, malaise<br>± Joint effusion<br>± Limp |
| **Differentials** | • **Bone malignancy**<br>• Crystal arthropathy<br>• Charcot joint<br>• Avascular necrosis<br>• RA<br>• Fracture<br>• Septic arthritis | • **Crystal arthropathy**<br>• **Cellulitis/bursitis**<br>• Viral arthritis<br>• Transient synovitis<br>• RA<br>• OA |
| **Investigations** | **Bloods:**<br>• **FBC:** ↑WCC, CRP, ESR<br>• **U&Es, LFTs, blood gases**<br>• **Urate:** indicate gout (septic gout)<br>**Blood culture (≥2):** positive<br>**X-ray:**<br>• Abnormal trabecular architecture<br>• Radiolucent/moth-eaten areas<br>• Osteopenia<br>• Periosteal thickening<br>**USS:** identify if coexistent joint effusion (septic arthritis)<br>**MRI: diagnostic** (shows if subperiosteal abscess)<br>**Bone scintigraphy:** ↑radionucleotide uptake<br>**Bone biopsy:** culture & sensitivity (at debridement) | **Bloods:**<br>• **FBC:** ↑WCC, CRP, ESR<br>• **U&Es, LFTs, blood gases** (may show signs of sepsis)<br>• **Urate:** indicate gout (can have septic gout)<br>**Blood culture (≥2):** positive<br>(± urine, sputum, wound culture)<br>**X-ray:** normal<br>(may be widened joint space / soft tissue swelling)<br>**Joint aspiration & culture: diagnostic**<br>(+ Gram stain, light microscopy)<br>**USS:** shows joint effusion & can guide aspiration<br>**MRI:** ↑soft tissue definition & shows if coexistent osteomyelitis |
| **Management** | 1. **IV ABX** if stable wait for culture results<br>• Minimum 6w (IV then switch to PO)<br>2. **Surgical decompression / aspiration**<br>→ Drain abscesses, debride infected tissues<br>→ **Remove prostheses**<br>3. **Supportive:**<br>• Analgesia, bed rest, immobilise/splint then physio<br>**= Need urgent (same day) referral to orthopaedics** | **= Medical emergency – immediate admission**<br>1. **Immediate resuscitation:** ABCDE / SEPSIS 6<br>2. **Immediate IV ABX** empirical until culture results<br>• IV then switch to 6w PO<br>3. **Joint drainage & washout (lavage)**<br>→ If deep-seated or Tx failure<br>→ If prosthesis in place do in orthopaedic theatre<br>4. **Supportive:**<br>• Analgesia, bed rest, immobilise/splint then physio |
| **Complications** | • Bone/cartilage necrosis<br>• Chronic infection<br>• Sepsis<br>• Limb deformity<br>• OA | |

## Discitis

*→ inflammation of intervertebral discs (often due to infection)*

**Risk factors:** IVDU, long-term steroids, immunosuppressed, autoimmune disorder, post-surgery

**Symptoms:** constant back pain & stiffness, tenderness over affected joint ± heat ± fever

**Investigations:** blood cultures, FBC, CRP/ESR, U&Es, LFTs, urine culture, X-ray/MRI

## Viral arthropathy

**Symptoms:**
- Viral prodrome ± characteristic rash
- Symmetrical small joint involvement (pain & inflammation)

**Investigations:**
- FBC, CRP, ESR
- LFTs, U&Es
- Serum antibodies (Hep B etc.)
- RF, anti-CCP, ANA (r/o other DDx)

**Management:** **self-limiting**
- **Supportive:** NSAIDs, bed rest

**Causes of viral arthropathy:**
- Parvovirus B19
- Hep B & C
- HIV
- Rubella
- EBV

**DDx of viral arthropathy:**
- RA
- Reactive arthritis
- SLE
- Rheumatic fever
- Ankylosing spondylitis

## Infections in elective orthopaedic surgery

**Culture from within joint = gold standard for Dx**

| | Early (<2w post-op) | Late (>2w post-op) |
|---|---|---|
| **Organisms** | *Staph. aureus* | Skin commensals (coag –ve staph, *Cutibacterium*) |
| **Management** | **DAIR** (debridement, ABX, implant retention)<br>• Debride, irrigate & send tissue for culture<br>• Exchange removable parts **but keep most**<br>• Combination of ≥2 ABX (e.g. ABX-loaded beads) | **1 or 2 stage revision surgery:**<br>• Remove prosthesis & send tissue for culture<br>• Tx infection with ABX (+ temporary cement spacer)<br>• Implant new prosthesis when infection cleared |

**PREVENTION:**

1. **Before surgery:**
   - Screen & decolonise MRSA / *Staph. aureus*
   - Optimise immunosuppressant medication
   - Eradicate any ongoing infections
2. **Peri-operative:**
   - **Prophylactic** ABX → 30min before incision Most important
     → Follow local guidelines e.g. flucloxacillin, cefuroxime, teicoplanin
   - Skin preparation → alcoholic chlorhexidine or iodine ± iodine-impregnated incision drapes
   - Laminar air flow in theatre
3. **Post-operative:**
   - Normal infection control (hand hygiene, ward cleaning etc.)

# Musculoskeletal malignancy

## Soft tissue swellings

| Benign | Malignant |
|---|---|
| <5cm or slow-growing<br>Painless<br>Superficial to fascia<br>Well-circumscribed<br>Homogeneous appearance | >5cm or rapid increase in size*<br>Painful*<br>Penetrates fascia (deep within muscle)*<br>Irregular border / not contained<br>Heterogeneous radiological appearance |
| e.g. lipoma, leiomyoma, schwannoma | e.g. sarcoma |

***Urgent referral if red flags:**

1. 2ww for USS
2. **If USS worrying/unclear:** 2ww cancer pathway (MRI & biopsy)

**If no worrying features:** observe or biopsy + histology

## Primary bone tumours

| | **Osteosarcoma** (most common) | **Chondrosarcoma** | **Ewing's sarcoma** |
|---|---|---|---|
| **Epidemiology** | → Children/adolescents (M>F)<br>→ 2nd peak in old age (in Paget's / post radiotherapy) | Adults >50y | Children/adolescents (rare) |
| **Common sites** | Distal femur, proximal tibia & humerus | **Flat bones:** pelvis, scapula, rib | **Diaphysis of bones** |
| **Associated Sx** | Lung mets | Pain, mechanical Sx, pathological fractures | Systemic upset (weight loss, fever, ↑ESR) |
| **Treatment** | 1. Chemotherapy<br>2. Surgery (+ more chemo) | **Not sensitive to chemo**<br>1. Surgery | 1. Chemotherapy<br>2. Surgery |
| **5y survival** | **No mets at Dx:** 60–65%<br>**Mets at Dx:** 20% | **Low grade:** 90%<br>**High grade:** 60% | 50% |

## Multiple myeloma

→ ***malignancy of plasma cells of bone marrow:*** *proliferation of* ***one type of plasma cell*** *producing* ***one type of Ig***

SYMPTOMS:

- Bone pain (commonly backache)
- Fractures of long bones & vertebral collapse
- **Hypercalcaemia** (due to increased osteolytic activity)
- Anaemia, neutropenia, thrombocytopenia
- ± Renal impairment & recurrent infections

**Average age:** >70y

**RF:** Afro-Caribbean

INVESTIGATIONS:

- **FBC:** anaemia, ↓WCC, ↑ESR/CRP
- **U&Es, LFTs:** deranged renal function in 20%
- **Bone profile:** hypercalcaemia
- **Plasma electrophoresis:** ↑monoclonal Igs
- **Urine microscopy:** Bence Jones proteins (monoclonal light chains)
- **X-rays:** punched-out osteolytic lesions ('pepper-pot')
- **Bone scintigraphy:** may have cold spots
- **Bone marrow biopsy:** >10% clonal plasma cells

**Poor prognosis:** 6y median survival

MANAGEMENT:

- **Supportive for complications** e.g. anaemia, renal failure
- **Localised disease:** radiotherapy
- **Widespread disease:** chemotherapy

## Metastatic bone disease

→ *most common cause of destructive bone disease in adults*

→ **70% in axial skeleton (mostly in spinal column)**

### SYMPTOMS:

- **Bone pain** (unremitting, dull ache → **night pain**)
- **Pathological fractures** (sudden worsening of pain)
- Hypercalcaemia
- Spinal cord compression
- **B Sx:** weight loss, night sweats

### ON EXAMINATION:

- Bony tenderness
- Swelling
- Reduced ROM in joints
- Lymphadenopathy
- Anaemia
- Neurological deficit

NB: if no Hx of cancer, consider primary bone tumour

### INVESTIGATIONS: find primary source

- **FBC:** anaemia, ↓WCC, ↑ESR/CRP
- **U&Es, LFTs, TFTs, bone profile:** hypercalcaemia
- **Tumour markers:** PSA for prostate cancer
- **Myeloma screen:** electrophoresis + urinalysis
- **X-rays:** lytic or sclerotic lesions
- **CT/MRI:** for primary tumour (chest, abdo, pelvis)
- **Bone scintigraphy:** hot spots
- **CXR**
- **CT-guided biopsy:** if uncertain of Dx

### MANAGEMENT:

1. **Find primary source:** X-ray, CT, MRI, bone scan
2. **Supportive**
    - Pain relief
    - Bone protection: splints/bisphosphonates (assess fracture risk)
3. **Chemo/radiotherapy**
4. **Surgery:**
    - Stabilisation if risk of fracture in long bones / vertebrae
    - Arthroplasty if joint involvement

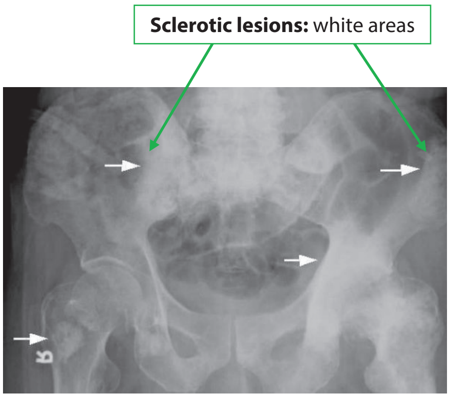


**Fig. 19.1**

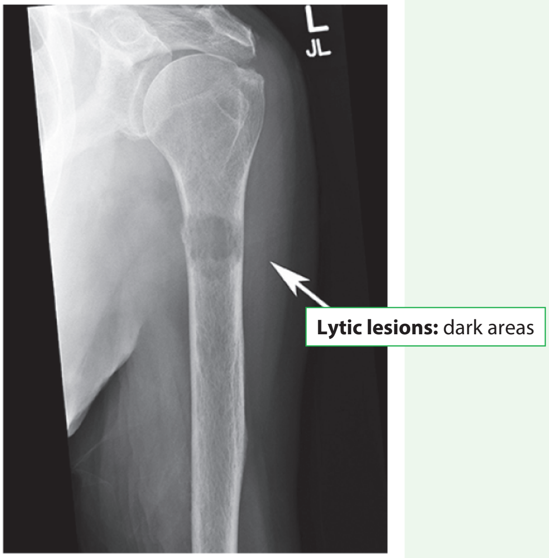


**Fig. 19.2**

**Common origins of bone mets:**
**PB-KTL ('lead kettle')**

| | |
|---|---|
| **P**rostate | **K**idneys |
| **B**reast | **T**hyroid |
| | **L**ung |

**Differentials:**

- Multiple myeloma
- Lymphoma
- Bone infection

# Diseases of the bone

## Osteomalacia (rickets in children)

*→ vitamin D deficiency causing **poor bone mineralisation** (but normal deposition & microarchitecture)*

**PRESENTATION:** often vague symptoms
- **Fragility fractures:** especially lower limb
- Vague bone/muscle pain/tenderness
- Proximal muscle weakness → waddling gait = **late sign**
- Signs of hypocalcaemia: tetany/spasm
- Costochondral swelling (chest pain/tenderness)
- **Deformities more common in kids:** bow legs / knock knees, Harrison sulcus, dental abnormalities, craniotabes

**INVESTIGATIONS:** determine cause & r/o DDx
- **History & examination**
- **Bloods:** FBC, ESR, U&Es, TFT, **vit D**, sex hormones
- **Bone profile:** Ca, $PO_4^-$, ALP, PTH
- **Myeloma screen:** if unsure
- **X-ray**
  - **Looser zones** – 'pseudo-fractures' of cortex
  - pathological fractures
  - chondrocalcinosis

**MANAGEMENT:**

**Lifestyle:**
- **Diet:** oily fish, fortified cereal
- **Sun exposure:** 15min 3× week

**Supplementation:**
- **Vitamin D:** 880 Units daily (+ loading bolus if very deficient)
- **Calcium:** 1500mg daily

**Causes of / risk factors for vitamin D deficiency:**
- Lack of sun exposure (institutionalised, BAME)
- Poor dietary intake
- Malabsorption (coeliac, gastrectomy)
- Renal disease (e.g. renal osteodystrophy)
- Medications (phenytoin, bisphosphonates)

**DDx of osteomalacia:**
- Osteoporosis
- Fibromyalgia
- Polymyalgia rheumatica
- Polymyositis
- RA
- Multiple myeloma
- Metastatic bone disease

## Paget's disease of bone

*→ **increased bone turnover** with excessive resorption followed by **rapid deposition of disorganised bone***

Paget's = 2nd most common bone disease after osteoporosis

**PRESENTATION:** usually asymptomatic
- **Bone pain** (localised in 1 or multiple sites)
- Pathological fractures
- Deformity: ↑ bone size, kyphosis, bowing

**COMPLICATIONS:**
- Headache
- Deafness
- Nerve root / cord compression
- Osteosarcoma (**rare**)

**INVESTIGATIONS:**
- **Bone profile:** raised ALP
- **Tests as above to r/o other bone disease**
- **X-ray:**
  - Thickened, coarse cortical bone
  - Mixed lytic & sclerotic lesions
  - **'Blade of grass sign'** in long bones (V-shaped lucency)
  - **Osteoporosis circumscripta** = large lytic lesions of skull

**MANAGEMENT:** serial ALP shows efficacy of Tx
- **Analgesia** – NSAIDs, paracetamol
- **Bisphosphonates** – ↓ bone turnover
- **Surgery** – if deformity, nerve compression

**→ Monitor for osteosarcoma**

**Risk factors for Paget's disease:**
- Age (rare <50y)
- Genetics

**Most commonly affected bones:**
- Pelvis
- Spine
- Skull
- Femur
- Sacrum
- Tibia

## Osteonecrosis

→ ***bone death*** *due to compromised blood supply*
→ marrow cells die within 12h – cortex spared as alternative blood supply from periosteum
→ new bone laid down (↑ mass on X-ray) followed by necrosis & eventually articular cartilage destruction (OA)

**PRESENTATION:** 1/3 are asymptomatic

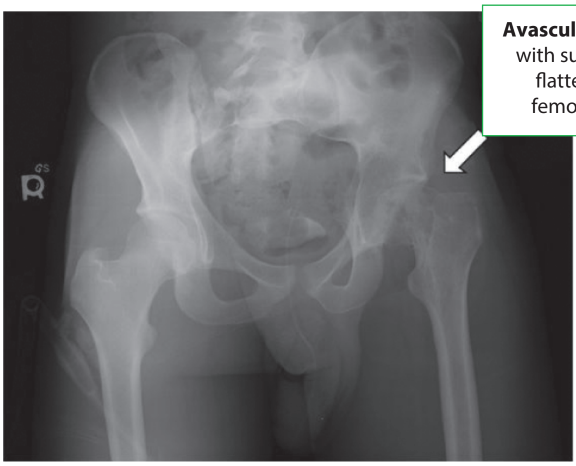
- **Pain** (near a joint & at rest)
- Stiffness / limited ROM

**DIAGNOSIS:**

→ X-ray (no changes seen for months)
→ MRI (shows changes earlier)
→ Radionucleotide imaging (↓ uptake)

**MANAGEMENT:**

1. Analgesia, avoid weight-bearing, splinting
2. Treat underlying cause
3. Surgery (e.g. bone decompression, arthroplasty)



**Avascular necrosis** with subsequent flattening of femoral head

**Fig. 19.3**

**Common sites of osteonecrosis:**

- Femoral head & condyles
- Humeral head
- Proximal tibia
- Proximal talus
- Proximal scaphoid

**Risk factors for osteonecrosis:**

- **Trauma/fracture**
- **Sickle cell disease**
- Steroids/Cushing's
- Bisphosphonates
- Autoimmune disorder (SLE, APS)
- Infection
- Malignancy
- Gaucher disease (congenital)

**Idiopathic conditions:**
Perthes' & osteochondritis dissecans

**Differentials:**

- Inflammatory synovitis
- Osteoarthritis
- Soft tissue trauma
- Bone malignancy

# Osteoarthritis

→ **Degenerative disorder of synovial joint**
→ Progressive loss of cartilage + subchondral bone remodelling

OA = most common type of arthritis

**Joints most commonly affected:**

**Weight-bearing: hips & knees** + spine, shoulder & PIPJs/DIPJs
**Usually asymmetrical**

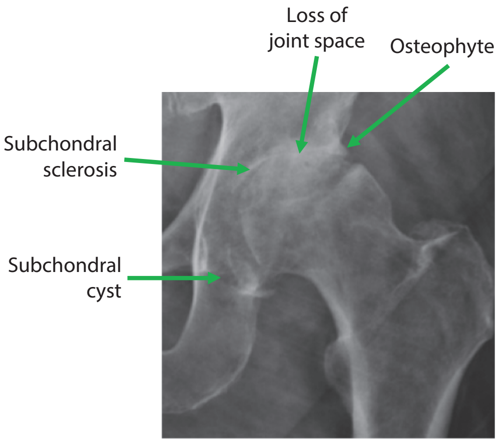


**Fig. 19.4** Radiographic features of osteoarthritis at the hip.

**Complications of arthroplasty:**

**Major complications:**

- Dislocation = 2–3% (risk highest in first 3m)
- Infection = 1% (remove implant & ABX)
- DVT/PE (can be fatal) = 0.3–0.9%
- Neurovascular = 0.3% (sciatic, femoral, brachial plexus)
- Wear & loosening → most **last 10–15y** (outlive patient) → try to delay in younger patients

**Minor complications:**

- Numbness along scar line
- Joint stiffening after sitting
- Clicking of components

## Pathology

Overall fibrillation & softening of articular cartilage → gradually erodes

## Risk factors

**Abnormal force** on normal cartilage **OR** normal force on **abnormal cartilage/bone:**

| Primary | Secondary |
|---|---|
| Age (25% >65y have Sx)<br>FHx/genetics<br>Female<br>Obesity<br>Abnormal joint loading | Trauma<br>Joint deformity (e.g. DDH, CAM deformity of hip)<br>Instability (hypermobile, Ehlers–Danlos)<br>Infection/inflammation (septic arthritis, RA, crystal arthropathy)<br>Bone/joint disorders (Paget's, Perthes', SUFE, DDH)<br>Metabolic/endocrine (thyroid, DM) |

## Clinical presentation

- **Pain – worse with movement, relieved with rest**
- Stiffness – if present in morning, **lasts <30min**

} affects functioning

## On examination

- **Pain** – on movement of joint / joint tenderness
- **Reduced ROM** & crepitus
- Swelling & deformity
- Muscle weakness / wasting around joint

## Investigations

OA = usually a clinical diagnosis

- Hx & examination
- **X-ray** – **don't always match clinical severity**
- MRI & blood tests – r/o other causes e.g. inflammatory, malignancy

**Radiographic findings:** → **'LOSS'**

- **L**oss of joint space
- **O**steophytes
- **S**ubchondral cysts
- **S**ubchondral sclerosis

## Management[1]

**Aim:** pain relief & improve functioning

1. **Lifestyle:**
    - Weight loss, adapting activities, appropriate footwear
    - Exercise & physiotherapy (swimming/cycling may be good)
2. **Pharmacological:**
    - **1st line:** paracetamol ± topical or PO NSAIDs → WHO ladder
    - **2nd line:** corticosteroid injections
3. **Surgery:** if significant pain despite analgesia or impacting ADLs & QoL (despite conservative Mx)
    - Arthroplasty (usually) → **counsel on risks, benefits, post-op side-effects**

**Post-op care:**

- Post-op exercise rehab & physio
- Follow up in 6–8w
- Restart driving & non-active jobs in 6w (active jobs in 12w)

Approx. 90% of hip & knee replacements are successful

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[1] NICE (2014, updated 2020) *Osteoarthritis* [CG177]

# Primary survey

**<C>ABCDE + continuous reassessment**

| | | |
|---|---|---|
| <C> | Catastrophic haemorrhage | **Manage:**<br>• Direct pressure until tourniquet can be applied (max. 30–60min of tourniquet → **get to theatre ASAP**) → document time applied |
| **A** | Airway<br>± C-spine control | **Assess:** added sounds (stridor/stertor), obstruction in mouth<br>**Manage:**<br>• Manoeuvres/adjuncts → jaw thrust only if C-spine damage<br>• Consider early intubation<br>• **Manual inline stabilisation** or immobilise with blocks & tape<br>• **CT scan ± MRI** if distracting injuries or low GCS |
| **B** | Breathing | **Assess:** work of breathing, RR, sats ($FiO_2$), ABG<br>**Manage:** $O_2$ if available (non-rebreather mask) |
| **C** | Circulation | **Assess:** HR, BP, central CRT, temperature of peripheries<br>**Manage:** 2 wide-bore cannulae<br>**If signs consistent with shock** (e.g. SBP <90)<br>**1.** Haemorrhage control<br>**2.** Tranexamic acid (TXA)<br>**3.** Fluid resuscitation (with blood products)<br>**4.** Keep warm<br>**No response to resuscitation = catastrophic haemorrhage** (often abdo/pelvic injuries)<br>→ **FAST scan** may be useful, X-ray if possible & transfer to theatre ASAP |
| **D** | Disability | **Assess:** AVPU/GCS, pupillary response, glucose (GCS <8 consider intubation) |
| **E** | Exposure | **Assess:** completely undress patient and thorough examination (keep them warm) |

*All trauma patients assumed to have C-spine injury until proven otherwise*

**Sources of bleeding:** 4 & floor
- Chest
- Abdomen
- Pelvis
- Long bones

# Secondary survey

**= exhaustive head-to-toe assessment of the patient's injuries**

## History

| | | |
|---|---|---|
| **A** | Allergies | To what & results |
| **M** | Medications | What, when, why? |
| **P** | Past medical Hx | Including surgery & injuries |
| **L** | Last oral intake | What, when? |
| **E** | Events leading to situation | MOI, precipitating cause |

Mechanism of injury (MOI)
= very important

## 1. Head examination

- GCS – deficit despite resuscitation?
- Skull fractures
- Eyes – orbital fractures, pupillary response
- Auditory canal – any blood?
- Examine all facial bones & in mouth

**→ Open skull fracture needs meningitis vaccine**

## 2. C-spine & neck examination

- Palpate for centrality & tenderness } CT if any concerns
- Neurological examination of upper limbs } CT if any concerns
- Examine for soft tissue injuries / subcutaneous emphysema (pneumothorax)
- Hoarseness may indicate **laryngeal trauma** → consider intubation

## 3. Chest examination

- Inspect, palpate, percuss, auscultate

## 4. Abdominal examination

**Liver, spleen, intestines at risk → penetrating wounds need exploratory laparotomy**

- Inspect, palpate, percuss, auscultate
  - Abdo (bruises, abrasions, Cullen's / Grey Turner's signs)
  - Pelvic structures (ASIS, pubic symphysis, sacrum & coccyx)
  - Genitalia
- **DRE:** sphincter tone, blood, prostate position
- Pelvic injuries need **stabilising via pelvic binder** → often associated with major internal haemorrhage

  ***Caution with catheterising in pelvic injury***

## 5. Musculoskeletal examination

- **All joints/bones:** dislocation, stability, fracture } stabilise/clean/treat as appropriate
- **All wounds:** assess & document } stabilise/clean/treat as appropriate
- **Neurovascular assessment:** document
- Assess for compartment syndrome
- **X-ray/CT for any injuries**

**Trauma blood panel:**

- FBC, U&Es, LFT
- Lipase/amylase
- Coag screen, X-match
- Beta-hCG (or urinary)

**Trauma imaging:**

**X-ray:** CXR (+ lateral C-spine if ? injury)

**Trauma CT:** ASAP
Head, chest, abdo, pelvis
(only if patient = stable)

**Indications for laparotomy:**
→ Peritonism
→ GI bleed / +ve FAST scan
→ Penetrating trauma

**Areas assessed by FAST** (Focused Assessment with Sonography in Trauma)

→ **detects free fluid (70–95% sensitive)**

1. Pericardium
2. Pelvis
3. **RUQ:** between diaphragm & liver
4. **LUQ:** between spleen & diaphragm + spleen & kidney

**Pros:** very quick, on-site, no contraindications
**Cons:** limited use if obese, SC emphysema, cannot tell cause of fluid, cannot inspect retroperitoneal structures or organs, **cannot rule out injury**

**Abdominal signs indicating FAST:**
→ Distension
→ Absent bowel sounds
→ Guarding / rebound tenderness

# Head injuries

**All unconscious head injuries have spinal fracture until proven otherwise!**

**Features of severe head injury:** → need CT head within 1h

- GCS <13 or <15 2h after injury
- Focal neuro deficit
- Signs of ↑ ICP
- Suspected skull fracture
- Post-traumatic seizure
- Vomiting >1 time

## Managing head injuries[2]

### INITIAL MANAGEMENT

1. **ABCDE**
2. **Hx** of event, intoxication, seizures etc.
3. **CT head** and C-spine radiology
4. **Monitor** – GCS, pupils, limb movements, RR, HR, BP, temperature, $O_2$ sats
5. **Consider transfer to neurosurgical unit –** if GCS ≤8 or need neurosurgical intervention

   → **Follow AAGBI guidelines for transfer:**
   - Stabilise patient prior to transfer (correct hypotension, intubate if GCS ≤8)
   - Ensure trained professional present with access to drugs which may be needed
   - Monitor obs throughout transfer

### DETERIORATING HEAD INJURIES

**Signs:**

→ **Decreasing GCS**
→ Development of **focal neuro signs**
→ **Pupil dilates** on side of haematoma
→ ± Reflexive bradycardia & hypertension (Cushing's sign)

**Management:**

**Urgent CT scan** & ICP management

## Base of skull fracture

From **severe traumatic head injury** & many have concurrent **facial / skull vault fractures**

### SYMPTOMS:

**Petrous bone #:** CSF otorrhoea, otorrhagia, Battle sign
**Anterior fossa #:** CSF rhinorrhoea, raccoon eyes sign

### COMPLICATIONS:

- CSF leak
- Intracranial haemorrhage / infection
- Cranial nerve palsies
- Sensorineural hearing loss
- Carotid injury

**MANAGEMENT: refer to neurosurgery**

**Signs of skull fracture:**

- Raccoon eyes, Battle sign
- CSF leak
- Otorrhoea/rhinorrhoea
- Cranial nerve palsy

**Only discharge head injuries if:**

- **GCS 15/15**
- Symptoms resolved
- No indication for CT
- Supervision at home

→ **Safety-net for dropping GCS / focal signs**

**Delayed complications of head injury:**

- Hypoxia, hypotension
- Oedema/hydrocephalus = raised ICP
- Ischaemia

**Intracranial pressure (ICP) management**

1. **Correct physiology** – manage hypoxia, hypercapnia, hypovolaemia
2. **Decrease metabolic rate** – sedate & intubate, prevent fever, treat seizures
3. **Pharmacological** – IV mannitol or hypertonic saline
4. **Surgical management** – haematoma evacuation / decompressive craniectomy

**Common causes of skull base fracture:**

- RTAs
- Sports injuries
- Falls
- Assault

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[2] NICE (2014, updated 2019) *Head injury* [CG176] & AAGBI (2019) *Guidelines for safe transfer of the brain-injured patient*

## Extradural haemorrhage

Bleeding of vessels causing haematoma **between skull & dura**
→ usually due to **temporal or parietal bone fracture** (trauma lateral to eye)
→ **damages middle meningeal artery**

**SYMPTOMS:** lucid interval of hours – days

- Reduced consciousness / GCS **delayed after trauma**
- Headache, N&V, confusion, seizures
- If untreated, raised ICP causes:
  - hemiparesis, brisk reflexes
  - dilated ipsilateral pupil
  - respiratory irregularities / apnoea

**INVESTIGATIONS:** CT/MRI head → do not do LP
→ **biconvex, well rounded, collection of blood** (limited by sutures)

**MANAGEMENT:** urgent referral to neurosurgery
→ Craniotomy/ burr hole ± vessel ligation
+ reverse any clotting abnormalities & treat cause of fall/trauma

## Subdural haemorrhage

Bleeding from **bridging veins** causing haematoma **between dura & arachnoid**
→ usually due to **low impact trauma** but this may be minor / far in the past, so not always remembered
→ **acute subdural haematoma** may occur in deceleration injuries & present with ↓GCS & **dilated/non-reactive pupil** *On ipsilateral side to haematoma*

**SYMPTOMS:**

- Fluctuating consciousness (lose it, then regain, then lose it)
- Headache / unsteadiness / personality change
- Seizures & focal neuro signs e.g. unequal pupils / hemiparesis

**INVESTIGATIONS:**

- CT/MRI head
  - **Acute:** homogeneous, hyperdense crescent-shaped collection of blood ('sickle-shaped')
  - **Subacute (10–14d):** isodense collection (harder to recognise)
  - **Chronic (>3w):** hypodense collection

± midline shift

**MANAGEMENT:** urgent neurosurgical referral

1. **Large / midline shift / neuro deficit:** craniotomy / burr hole
2. **Otherwise:** imaging and monitoring may be adequate

+ reverse any clotting abnormalities & treat cause of fall/trauma

**DDx of extradural haemorrhage:**

- Epilepsy
- Carotid dissection
- CO poisoning

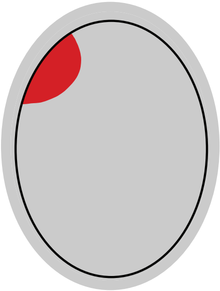

**Fig. 19.5** Diagrammatic representation of CT head showing biconvex extradural haemorrhage.

**DDx of subdural haemorrhage:**

- Stroke
- Dementia/delirium
- CNS mass (tumour/abscess)

**Risk factors for subdural haemorrhage:**

- Elderly (weak veins)
- Anticoagulation
- Falls (epileptics/alcoholics)

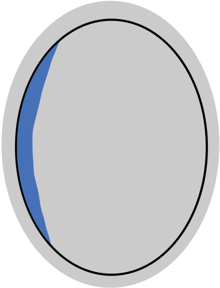

**Fig. 19.6** Diagrammatic representation of CT head showing crescent-shaped subdural haemorrhage.

## Subarachnoid haemorrhage

### CLINICAL PRESENTATION:

- **Thunderclap headache** – severe & sudden
- **Vomiting & LOC**

### ON EXAMINATION:

- **Meningism** – stiff neck, photophobia, +ve Kernig's
- **Subhyaloid (intraocular) haemorrhage**
- **Reduced GCS** (not always)

### INVESTIGATIONS[3]:

1. **Non-contrast CT:** detects >95% SAH within first 24h

If CT is negative = consider **lumbar puncture**

→ Look for xanthochromia
→ Must be ≥12h after headache onset

2. **Bloods:** FBC, ESR, U&Es, glucose, LFT, clotting

### MANAGEMENT[3]:

1. **Refer to neurosurgery:** investigate for cause e.g. berry aneurysms
2. **Nimodipine:** calcium antagonist to prevent vasospasm
3. **Radiological intervention:** clipping/coiling of berry aneurysms

**→ maintain hydration but ensure SPB <180**
**→ anti-epileptic medication if seizures present**

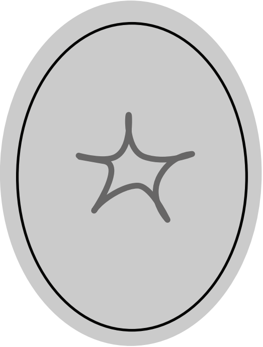

**Fig. 19.7** Diagrammatic representation of CT head showing star-shaped SAH.

**Causes of SAH:**

- Berry aneurysms = 80%
- AVMs = 15%
- Intracranial malignancy

**Risk factors for SAH:**

- PKD, CTDs (associate with berry aneurysms)
- **Uncontrolled HTN**
- **Anticoagulants/coagulopathies**
- Alcohol/smoking

**Differentials of SAH:**

- Migraine
- Meningitis
- Coital headache – much briefer (30–60min) & recurrent
- Reversible vasoconstriction syndrome
  → after decongestants
- Intracerebral bleed
- Cortical vein thrombosis

**Complications of SAH:**

- Death (30%)
- Rebleed (20% in first 3d)
- Hydrocephalus
- Cerebral vasospasm → ischaemia
- Hyponatraemia

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[3] NICE Draft Guideline (2021) *Subarachnoid haemorrhage due to ruptured aneurysms* [GID-NG10097]

# Spinal cord injury

→ Direct damage or 2° to pressure

Classified using ASIA chart[4]

## Types of spinal cord injury

| Complete | Incomplete |
|---|---|
| **No motor or sensory function below injury level**<br>• Bulbocavernosus reflex still present | **Some motor or sensory function below injury level**<br>• Voluntary anal contraction **or**<br>• Palpable/visible muscle contraction **or**<br>• Perianal sensation |

## Acute phase conditions (resulting from spinal cord injury)

| | Neurogenic shock | Spinal shock |
|---|---|---|
| **Pathogenesis** | Loss of autonomic tone causing **haemodynamic changes**<br>→ type of distributive shock | Loss of spinal reflexes causing **flaccid areflexia & paralysis**<br>→ not true form of shock |
| **Symptoms** | **Occurs if injury above T5**<br>• Hypotension & bradycardia | Can occur with **any cord injury**<br>• Hypotension & bradycardia<br>• **Absent reflexes (including bulbocavernosus)** |
| **Management** | **Lasts 3d – 3w**<br>• Vasoactive drugs & close monitoring | **Lasts weeks to months**<br>• Bladder & bowel management<br>• Monitor for respiratory difficulty<br>• Monitor for fever (cannot perspire where paralysed) |

## Spinal cord anatomy

**Descending tracts:** motor tracts

Tracts to upper limbs = medial
Tracts to lower limbs = lateral

→ **corticospinal** = voluntary movement of ipsilateral limbs
→ mainly in **posterior half** of spinal cord (except anterior corticospinal)
→ most **decussate in brainstem** = injury causes ipsilateral paralysis

**Ascending tracts:** sensory tracts

Tracts to upper limbs = lateral
Tracts to lower limbs = medial

→ **DCML** = posterior half of spinal cord
→ **Spinothalamic** = anterior half of spinal cord
→ **DCML** decussates in brainstem = injury causes **ipsilateral** loss of **fine touch, vibration & proprioception**
→ **Spinothalamic** decussate 2–3 levels above where they enter cord = injury causes **contralateral pain & temperature loss**

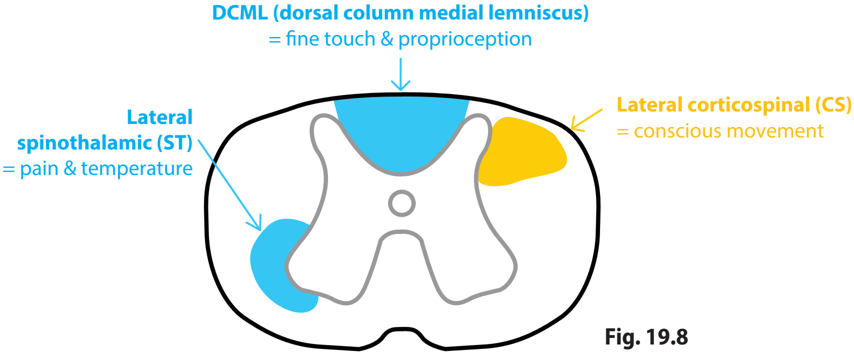
DCML (dorsal column medial lemniscus)
= fine touch & proprioception
Lateral spinothalamic (ST)
= pain & temperature
Lateral corticospinal (CS)
= conscious movement

**Fig. 19.8**

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[4] ASIA (2011) International standards for neurological classification of spinal cord injury. *J Spinal Cord Med*, 34:535

## Clinical syndromes

**BROWN-SÉQUARD SYNDROME** = hemisection of the spinal cord

- Loss of **ipsilateral motor & fine touch / proprioception** below level injured
- Loss of **contralateral temp / pain** 2–3 levels below level injured

**Cause:** penetrating trauma e.g. gunshot wound

**CENTRAL CORD SYNDROME** = injury to central grey matter of cord

- Loss of motor function in **upper limbs > lower limbs** (**medial** motor tracts affected)
- Variable sensory loss

**Cause:** pre-existing cervical spondylosis (elderly) + hyperextension injury → causes simultaneous anterior & posterior cord compression

**ANTERIOR CORD SYNDROME** = injury to anterior part of cord

- **Pain & temperature bilaterally** affected
- Loss of motor function in **lower limbs > upper limbs** (**lateral** motor tracts affected)
- **Sparing** of fine touch & proprioception (dorsal columns)

**Cause:**

1. Ischaemic damage via injury of anterior spinal artery
2. **Burst fracture:** crushed vertebral body pushed posteriorly into cord

**POSTERIOR CORD SYNDROME** = injury to posterior part of cord

- **Fine touch & proprioception** lost **below damage** (bilateral if both columns affected)
- **Sparing** of motor function

**Cause:**

1. Neck hyperflexion
2. B12 deficiency
3. Posterior spinal artery infarct

## Evaluation & management of spinal cord injury[5]

**IMMEDIATE:** ATLS approach

1. **Triple immobilised:** manual inline stabilisation, back board for transport
2. **Consider intubation:** if injury **above C5** (potential for airway compromise)
3. **Pain management**

**ON ADMISSION:**

1. **C-spine imaging:** CT C-spine, X-ray spinal column
2. **Prophylaxis:** VTE, pressure sore prevention
3. **Monitor:** BP, urine output & bowel function **and neuro obs**

**DEFINITIVE:**

1. **Reduce pressure / decompress cord:** e.g. remove osteophytes etc.
2. **Stabilise** – surgical management + soft collars
3. **Rehab** – physio, SALT → set realistic goals with patient

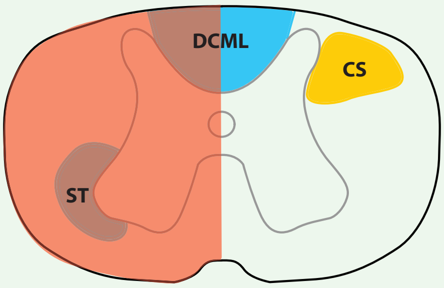

**Fig. 19.9** Area of spinal cord damaged in Brown-Séquard syndrome.

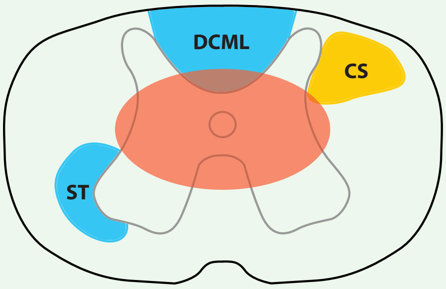

**Fig. 19.10** Area of spinal cord damaged in central cord syndrome.

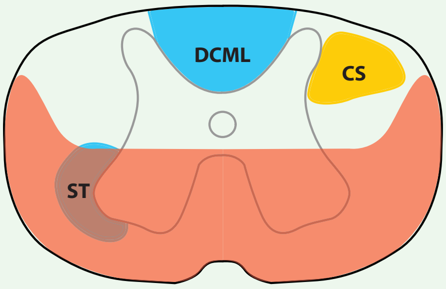

**Fig. 19.11** Area of spinal cord damaged in anterior cord syndrome.

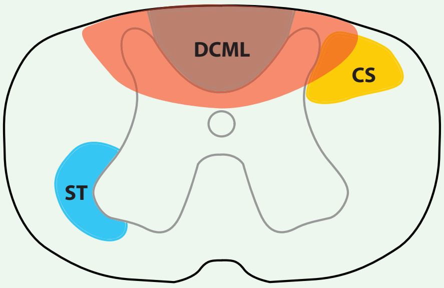

**Fig. 19.12** Area of spinal cord damaged in posterior cord syndrome.

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[5] NICE (2016) *Spinal injury* [NG41]

# Chest injuries

## Potentially life-threatening chest injuries

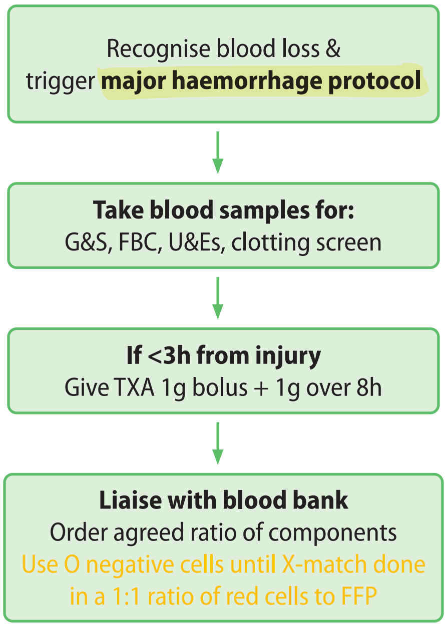
**Remember with 'ATOM FC'**

| | | |
|---|---|---|
| **A** | **Airway obstruction** | **Symptoms:**<br>• Stridor/gurgling<br>• Hoarse voice<br>• Surgical emphysema<br>**Management:**<br>**1.** Manoeuvres<br>**2.** Suction<br>**3.** Adjuncts<br>**4.** Intubation |
| **T** | **Tension pneumothorax** | **Symptoms:**<br>• Trachea deviates away from pneumothorax<br>• ↓Breath sounds<br>• Hyper-resonant percussion<br>**Management:**<br>**1.** Needle thoracotomy + chest drain |
| **O** | **Open pneumothorax** | **Symptoms:**<br>• Penetrating chest wall injury (open wound)<br>• + Clinical signs of pneumothorax (as above)<br>**Management:**<br>**1.** Partially occlusive (3 sides sealed) dressing + chest drain<br>**2.** Surgical closure |
| **M** | **Massive haemothorax**<br>**Blood loss >1500ml or >200ml/h** | **Symptoms:**<br>• Dull percussion<br>• ↓Breath sounds<br>• CXR consolidation<br>• **Haemorrhagic shock**<br>**Management:**<br>**1.** Fluid/haemostatic resuscitation<br>**2.** Chest drain → chest thoracotomy if severe |
| **F** | **Flail chest**<br>**≥2 contiguous ribs fractured in ≥2 places** | **Symptoms:**<br>• Flail segment of chest wall<br>• Paradoxical chest movements<br>• Chest pain<br>**Management:**<br>**1.** Analgesia (± regional anaesthesia)<br>**2.** Close respiratory monitoring<br>**3.** ± Surgical fixation |
| **C** | **Cardiac tamponade** | **Symptoms:**<br>• Beck's triad<br>• **Shock**<br>**1.** **D**istended neck veins<br>**2.** **D**istant heart sounds<br>**3.** **D**ecreased BP<br>**Management:**<br>**1.** Fluid resuscitation<br>**2.** Pericardiocentesis |

# Major haemorrhage[6]

Major haemorrhage is 50% volume blood loss in 3h (practically, we class it as any bleeding causing HR>110 ± SBP<90)

## MANAGEMENT:



Recognise blood loss & trigger **major haemorrhage protocol**

**Take blood samples for:**
G&S, FBC, U&Es, clotting screen

**If <3h from injury**
Give TXA 1g bolus + 1g over 8h

**Liaise with blood bank**
Order agreed ratio of components
Use O negative cells until X-match done in a 1:1 ratio of red cells to FFP

**Fig. 19.13**

NHS guidelines suggest all hospitals have their own major haemorrhage protocol policy

**Indications for blood components:**

**Falling Hb:** RBCs
**APPT ratio >1.5:** FFP
**Fibrinogen <1.5:** cryoprecipitate
**Platelets <50 × $10^9$:** platelets

---

[6] Trust Policy for blood transfusion and major haemorrhage (Queens Hospital Burton)

# Burns

Damage to tissues causing necrosis and a hypermetabolic, inflammatory response

## Pathophysiology

**Local:** coagulation, ischaemia & necrosis **plus** cytokine release → inflammatory response (hyperaemia, erythema, oedema)

**Widespread:** **fluid loss** – hypovolaemia → distributive shock → systemic dysregulation (e.g. AKI, hypothermia, immunosuppression, ARDS, infection)

**Shock occurs when:**

- >15% SA involved (>10% in children)
- if 50–70% SA involved = can be lethal

## Categories of burns

**Thermal:** (most common)

- Direct contact with hot object / flame (burns), or liquids/vapour (scalds)
- Damage depends on temperature, duration of exposure, thickness of skin

**Electrical:**

- Entry and exit burns visible superficially **but consider damage along internal path as well** e.g. vessel thrombus & rhabdomyolysis (leading to AKI)
- Severity depends on voltage and duration

**Chemical:** immediate, copious irrigation with water

- Difficult to remove source and often cause continuing damage
- Damage depends on substance (alkali worse than acid), concentration and exposure duration

**Cold (frostbite):**

- Freezing of water within tissues

**Irradiation:**

- Due to UV exposure (e.g. sunburn)

**Investigations**

- $O_2$ sats, ABG
- Serum electrolytes (↑$K^+$ & ↓$Na^+$)
- U&Es & creatinine (for AKI)
- FBC, serum protein, albumin

**If inhalation:** CXR, carboxyhaemoglobin levels

**Other types of burn:**

1. **Inhalation:** facial burns, hoarseness, stridor, soot in mouth/nose, airway oedema and obstruction
   → ***Consider inhaled toxic gases*** *e.g. CO*
   → 100% $O_2$ & consider intubation if airway swelling
2. **Circumferential:** full thickness burns around extremities causing restrictive **eschar (necrotic tissue)**
   → Results in compartment syndrome and acute limb ischaemia (check colour, temp, neurovascular status of limb)
   → Need **escharotomy** to release restriction

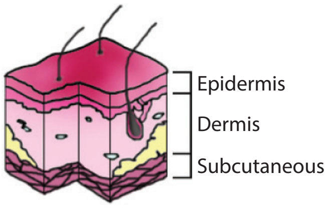
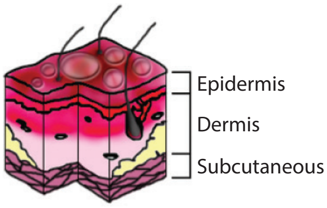
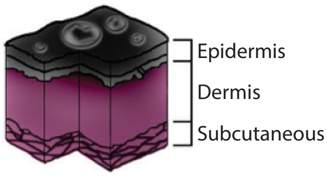
## Complications

| Early | Late |
|---|---|
| • **Hypovolaemia & shock** (↓BP, ↓UO) → AKI<br>• **Wound sepsis**<br>→ *S. pyogenes* in 1st week<br>→ *Pseudomonas* afterwards<br><br>• **Compartment syndrome / acute limb ischaemia** in circumferential burns | • Infection (± sepsis)<br>• DIC<br>• ARDS<br>• Curling's gastric ulcer (needs PPI prophylaxis)<br><br>• **Hypermetabolic state** (5–7d later)<br>→ Needs ↑ nutrition<br>→ ± Anabolic steroids ± beta-blockers |

## Assessing burn severity[7]

### 1. DEPTH

Fig. 19.14

| Degree | | Thickness | Characteristics | Appearance |
|---|---|---|---|---|
| 1st | | **Superficial:** epidermis | → **Pain**, erythema, swelling<br>→ Blanches |  |
| 2nd | 2a | **Superficial partial:** upper dermis | → **Pain**, erythema, vesicles/blisters<br>→ Blanches |  |
| | 2b | **Deep partial:** lower dermis | → **Minimal pain**, mottled, vesicles/blisters<br>→ **Non-blanching** | |
| 3rd | | **Full thickness:** subcutaneous tissue | → **No pain**, white, necrosis<br>→ Non-blanching<br>Heals with dense scar tissue (needs graft) |  |
| 4th | | **Most severe:** full thickness that may involve tendons, muscles, bones | | |

### 2. SURFACE AREA

- **Lund & Browder charts[8]:** determine % body SA affected
- Can estimate % SA with **rule of 9s** (palm of hand = approx 1%)

| Segment | % SA in adult |
|---|---|
| **Head** | 9% |
| **Trunk** | 36% (4 × 9%) |
| **Arms** | 18% (2 × 9%) |
| **Thighs** | 18% (2 × 9%) |
| **Lower legs and feet** | 18% (2 × 9%) |
| **Genital region** | 1% |

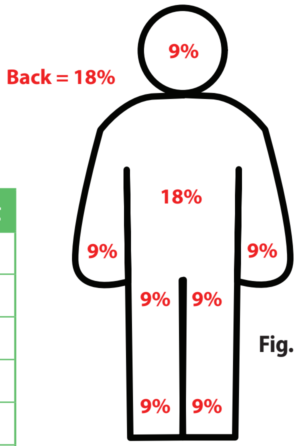


**Fig. 19.15** Rule of 9s.

**Burns first aid:**
Cool water 20min + cling film

## Management

1. **ATLS protocol** – primary & secondary survey + **burn cooling** (cool water)
2. **IV fluid resuscitation:** if burns >15% body SA (>10% in children)
   - **Parkland formula:** used to calculate volume of Hartmann's needed in first 24h
3. **Analgesia:** IV morphine
4. **Burn care:**

**1st & 2nd degree:** irrigation, topical moisturisers/antiseptics, dressings
**3rd & 4th degree:** early debridement of necrotic tissue, topical ABX, **skin grafts**

5. **Good nutrition**

**Systemic ABX:** if systemic infection

**Indications for referral[9]:**

- >15% of total body SA
- Deep dermal burns >5% body SA
- Full thickness burns
- Hands, face, feet, genitalia
- Circumferential injury
- Associated inhalation injury
- Chemical or electrical injuries
- Extremes of age
- Suspected non-accidental injury

**Parkland formula:**
**= 4 × total % SA × weight**
(give ½ in first 8h, then ½ in next 16h)
NB: new guidelines suggest only 2 × SA × weight due to risks of fluid overload

**Skin grafts:**

- Within 5d
- Must be infection-free
- Autograft = best option
- Cover donor site with dressing

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[7] McCaughey P, McAllister S (2016) Initial assessment and management of burns. *BMJ*, 352:h5583
[8] Lund C, Browder NC (1944) The estimation of areas of burns. *Surg Gynecol Obstet*, 79:352
[9] NICE (2020) CKS – *Burns and scalds, Scenario: First aid and initial management*

# General fracture principles

## Classification of fractures

OPEN FRACTURES → **Gustilo–Anderson classification**

| Type I | Type II | Type IIIa | Type IIIb | Type IIIc |
|---|---|---|---|---|
| - Skin **wound <1cm**<br>- Clean<br>- Simple fracture pattern | - Skin **wound ≥1cm**<br>- Moderate soft tissue damage<br>- Minimal comminution | **Extensive soft tissue damage** (still **adequate cover**)<br>**Or** multi-fragmented/segmental<br>**Or** crush / farm injury<br>**Or** massively contaminated | Type IIIa + **inadequate soft tissue cover**<br>(needs flap) | Type IIIa + **vascular injury**<br>(needs repair) |
| **Low impact** | **Moderate impact** | **High impact** | | |
| **Management** | | | | |
| Sutures often enough | | IV ABX, irrigation & surgical debridement<br>Reconstruction (**or** temporary external fixation **or** amputation) | | |

Increasing risk of **infection, non-union & amputation**

CLOSED FRACTURES → **Oestern–Tscherne classification**

| Grade 0 | Grade 1 | Grade 2 | Grade 3 |
|---|---|---|---|
| - Minimal soft tissue damage<br>- **Simple** fracture pattern | - **Superficial** abrasion/contusion<br>- **Mild** fracture pattern | - **Deep** abrasion<br>- Skin/**muscle contusion**<br>- **Severe** fracture pattern | - **Extensive** skin contusion<br>- Severe **damage to muscle**<br>- **Compartment syndrome** |
| **Indirect injury** | | **Direct injury** | |

## Describing fractures

1. Demographics – name, age, date, anatomical markers, projection
2. Anatomical site – name, side, bone & joint → proximal, middle, distal **or** diaphysis, epiphysis, metaphysis
3. Intra- or extra-articular
4. Fracture configuration – oblique, spiral, wedge etc.
5. Displacement – how distal fragment has moved relative to proximal AND proportion of surfaces in contact (e.g. 50%)
6. Angulation – describe in degrees & whether varus or valgus **→ need 2 views**
7. Shortening & rotation
8. Open/closed → associated soft tissue injury & neurovascular status

*TIP: start with describing the most obvious abnormality*

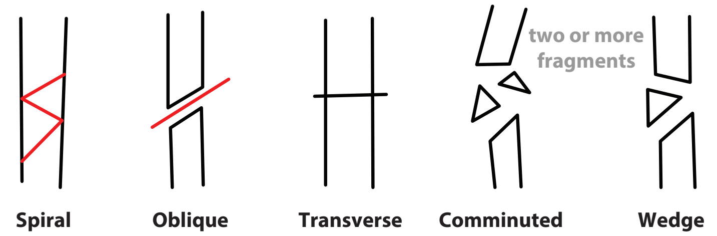

**Fig. 19.16** Fracture configurations.

**Exam tip:**
Always ask for a **2nd view** when given an X-ray

## Fracture healing

**1. Primary healing:** occurs if fragments can be reduced so **no fracture gap** via compression across the fracture
= **needs absolute stability** → important for fracture at **joint line** to enable a perfect healing position

**2. Secondary healing:** occurs if **fracture gap** with **some, controlled, motion** between fragments (e.g. using plates/nails)
= **needs relative stability** → if fracture fixed too rigidly **no movement at all** = won't allow callus formation

**Week 1:**
**Haematoma & inflammation**
Haematoma converted to **granulation tissue** by infiltrating cells

→

**Week 1–4:**
**Soft callus**
Deposition of **collagen & fibrocartilage** converts granulation tissue to soft callus

→

**Month 1–4:**
**Hard callus**
Calcified soft callus resorbed
Osteoblasts lay down matrix to form **woven bone**

→

**Year 1–2:**
**Remodelling**
Conversion to **lamellar bone** & medullary canal reforms

### FACTORS AFFECTING FRACTURE HEALING:

| Local | Systemic |
|---|---|
| Soft tissue trauma | Smoking, diabetes, PVD |
| Neurovascular injury | Nutrition |
| Bone loss | Age |
| Immobilisation | Previous irradiation |
| Infection/tumour | Drugs (persistent NSAIDs) |
| Interposition of soft tissues | Hormones |
| Bone type & site | Associated head injury |

**Healing at different sites:**
**3 weeks:** metacarpals
**6 weeks:** clavicle, radius, fibula
**10 weeks:** humerus
**16 weeks:** femur, tibia

**Delayed union:** fracture not healed within double the average healing time for that patient

**Non-union:** fracture not healed after double the average healing time for that patient **or** no evidence of progression on serial X-rays
→ Hypertrophic non-union e.g. not sufficiently stabilised
→ Atrophic non-union e.g. insufficient blood supply

### COMPLICATIONS OF BONE FRACTURES:

| **Immediate** (when broken) | **Acute** (during treatment) | **Long-term** |
|---|---|---|
| • Severe haemorrhage & shock<br>• Injury to surrounding organs<br>• Injury to surrounding nerves/vessels**<br>• Skin loss/damage | • Infection – debridement & ABX<br>• Fat embolism syndrome**<br>• Crush syndrome**<br>• Compartment syndrome**<br>**Immobilisation:**<br>• DVT & PE – give LMWH/aspirin<br>• Renal calculi / urinary retention<br>• Chest infections – esp. elderly<br>• Pressure sores & muscle wasting | • Non-union/mal-union<br>• Joint stiffness & contracture<br>• Osteoarthritis (if joint damage)<br>• Regional pain syndrome**<br>• Heterotrophic ossification<br>• Avascular necrosis<br>► head of femur<br>► lunate<br>► body talus<br>► proximal scaphoid |

****See below for more on these complications**

### NEUROLOGICAL DAMAGE ASSOCIATED WITH COMMON FRACTURES

| Fracture/dislocation | Nerve injury |
|---|---|
| Shoulder dislocation | Axillary nerve |
| Humeral shaft | Radial nerve |
| Supracondylar humerus | Anterior interosseus / median |
| Monteggia fracture | Posterior interosseus / radial |
| Distal radius | Median nerve |
| Posterior hip dislocation | Sciatic nerve |
| Knee dislocation | Common peroneal nerve |

## Fracture complications

### COMPARTMENT SYNDROME:

**Aetiology:** muscle swells in restricted fascial compartment → occludes arterial & venous flow → **ischaemia** → **necrosis**

**Causes:** follows trauma, fracture, tight casts/dressings, IV infusion

**Signs:**

- **Out of proportion pain** despite analgesia (**unremitting throbbing** that develops hours after injury)
- Pain on passive flexion/extension of fingers/toes of affected limb

**Investigations:** assess for swelling, neurovascular status of limb*, **passive stretch tests**

*Present pulse does not r/o this Dx

**Diagnosis:** measure compartment pressure

**Compartment pressure diagnostic if:**
>30mmHg **or**
<30mmHg difference from diastolic BP

**Management:** **surgical emergency**

1. Elevate limb, remove dressings / split casts open
2. Prompt fasciotomy

Calf & forearm = most common locations

### CRUSH SYNDROME:

**Aetiology:** systemic symptoms resulting from toxins released from crushed muscle tissues

**Causes:** traumatic rhabdomyolysis or prolonged fixed position

**Signs:** muscle pains, vomiting, confusion

- Hypovolaemic shock
- **AKI**
- **Hyperkalaemia**
- DIC
- **Raised CK**
- Metabolic acidosis

**Management:** **ABCDE**

1. Treat metabolic abnormalities & **catheterise** (IV fluids or dialysis)
2. Crushed limbs may need amputation

### FAT EMBOLISM SYNDROME:

**Aetiology:** lipid emboli circulate to lung capillaries → lodge & cause inflammatory response

**Causes:** 1–3d after long bone fracture → **rare!**

**Signs:**

- **Major:** petechial rash, confusion, **respiratory distress** (ground glass CXR)
- **Minor:** fever, tachycardia, oligo-/anuria
- **Haematological:** thrombocytopenia, anaemia

**Prevention:** early fixation & immobilisation of fractures

**Management:** supportive & Tx of organ dysfunction

### COMPLEX REGIONAL PAIN SYNDROME (CRPS):

**Aetiology:** dysregulation of central & autonomic nervous system → worsens with time

**Signs:**

- Allodynia/hyperalgesia (increased pain sensitivity)
- Swelling & skin changes (warmth, erythema & sweating)
- Motor dysfunction (weakness, tremor, ↓ ROM)

**Management:** **self-limiting** but can take up to 2y → may need regional nerve blocks

## Fracture management principles

**Always start with A to E / ATLS assessment**

Fracture may be a distracting injury

1. **Analgesia:** following WHO analgesic ladder
2. **Assess for:** **document all findings**
    - Open wounds / skin condition (swab open wounds)
    - Nerve damage (motor & sensory function)
    - Arterial damage (pulses, CRT, limb/extremity condition)
    - Compartment syndrome
3. **Order radiographs** (at least 2 views & include joint above & below)
4. **Provisional reduction** if closed fracture
5. **Fix/repair:**
    - Splint/cast (may be sufficient if initial reduction achieves good position)
    - External fixation device may be needed as temporary measure before surgery (allow swelling to reduce)
    - Definitive surgery (internal or external fixation)
6. **Repeat X-ray after intervention & recheck neurovascular status**

### FRACTURE FIXATION OPTIONS:

| Intermedullary nail | Lag screws ± plates | External fixation device |
|---|---|---|
| If fracture gap<br>Especially for midshaft fractures | If fracture gap can be closed<br>Especially fractures close to joints | If extensive soft tissue damage or contamination<br>Especially high energy injuries |

### SOFT TISSUE DAMAGE OPTIONS:

**1. Leave to heal by secondary intention:**

→ Wound edges not opposed, granulation tissue, contraction & scarring

**2. Primary closure (e.g. sutures) to allow healing by primary intention:**

→ Wound edges opposed, heals with minimal scar tissue

**3. Delayed primary closure:**

→ e.g. if wound is contaminated → leave 5–10d then close

**4. Skin grafts:**

→ Blocks of tissue (skin) from another area **without integral blood supply**

→ Need to be **placed in area of good blood supply**

**5. Skin flaps:**

→ Blocks of tissue **with integral blood supply** (fascia, fat, skin ± muscle)

- **local flap:** adjacent tissue stretched over defect
- **regional/pedicled flap:** tissue taken from further away but connected to original blood supply
- **free tissue flap:** tissue detached completely & reconnected to blood supply at site of defect

**Additional management for open fractures:**

1. **IV ABX** (within 1h of injury)
2. Check **tetanus status** – IV Ig if unvaccinated
3. **Irrigation** – saline-soaked gauze
4. **Splint** while awaiting theatre
5. **Surgical debridement** + fixation

→ Consider taking photos of wound

**Simple approach to fracture Mx: 4 Rs**

1. Resuscitate
2. Reduce
3. Retain (immobilise)
4. Rehabilitate

**Topical negative pressure therapy:**

Dressing + plastic wrap → pump sucks out air to provide **temporary wound closure / splinting**

**Benefits:** pulls edges together, removes excess fluid, ↓s contamination

# Pathological fractures

= fracture without significant trauma

**Risk factors for osteoporosis:**
- Age/female
- Previous fractures
- Parent fractured hip
- **S**teroids/**s**moking
- **H**yperthyroidism/**h**ypoparathyroidism
- **A**lcohol (>3 units/day)
- **T**hin
- **T**estosterone low
- **E**arly menopause (<45y)
- **R**enal/liver failure
- **E**rosive bone disease (RA/myeloma)
- **D**iet low Ca / vit D (BAME background)

SHATTERED

| Total score | Fracture risk | Recommendation |
|---|---|---|
| **≥9** | 33–100% | Prophylactic fixation |
| **8** | 15% | Clinical judgement |
| **≤7** | <4% | Observation ± other therapy |

**FRAX score parameters:**
- Age, gender, BMI
- Smoking/alcohol history
- Steroid use
- RA
- Fracture history

**DEXA scores:**

**T-score** = SDs below average for **young adult male**

**Z-score** = SDs below average for **age-matched control**

**Osteoporosis:** T-score **>2.5 SDs below** average

**Osteopenia:** T-score **1–2.5 SDs below** average

| Bone profile | Ca$^{2+}$ | PO$_4^-$ | ALP | PTH | |
|---|---|---|---|---|---|
| **Osteoporosis** | N | N | N | N | ↓BMD |
| **Osteomalacia** | ↓ | ↓ | ↑ | ↑ | Soft bones |
| **Paget's disease** | N | N | ↑ | N | Abnormal bone |
| **1° Hyperparathyroid** | ↑ | ↓ | ↑ | ↑ | Soft bones |

**Bisphosphonates:**
- Tablets or injections once a week
- Swallow with water on empty stomach
- Sit upright for 30min after

SE: GI upset, oesophageal ulcers, jaw necrosis

## Types

1. **Stress fractures:** normal bone*, but repetitive strain
2. **Loss of bone:** osteoporosis (especially NOF and vertebral fracture)
3. **Abnormal bone:**
   - Osteomalacia (often small, undisplaced fractures)
   - Paget's disease
   - Primary tumour (giant cell, myeloma, osteosarcoma)
   - Metastases (breast, lungs, thyroid, kidney, prostate)
   - Bone cysts
   - Congenital bone disorders e.g. osteogenesis imperfecta

*X-ray may only show periosteal reaction

## Management

1. **Orthopaedic referral** + **Mirels' score:** determine likelihood that a pathological lesion will result in fracture

| Points scored | 1 | 2 | 3 |
|---|---|---|---|
| **Site** | Upper limb | Lower limb | Trochanteric |
| **Pain** | Mild | Moderate | Functional |
| **X-ray appearance** | Blastic | Mixed | Lytic |
| **Size of lesion** | <1/3 cortex | 1/3–2/3 cortex | >2/3 cortex |

2. **Search for other mets:** CT spine / long bones / pelvis etc.
3. **Search for primary:** CXR, DRE, thyroid exam / USS, kidney USS

## Osteoporosis

→ ***reduced bone mass*** *& abnormal microarchitecture resulting in skeletal fragility & increased risk of fracture*

→ *the result of low peak bone mass &/or bone resorption > formation*

**PRESENTATION:** often asymptomatic until fragility fracture
- **Fragility fractures:** vertebral crush, NOF, wrist (FOOSH)
- **Loss of height / kyphosis / back pain**

**INVESTIGATIONS:**

Must r/o other causes of fragility fractures e.g. malignancy
- **History & examination → calculate FRAX score**
- **Bloods:** FBC, ESR, U&Es, TFT, vit D, sex hormones
- **Bone profile:** Ca$^{2+}$, PO$_4^-$, ALP*, PTH
- **Myeloma screen:** ESR, electrophoresis, Bence Jones protein
- **X-ray** – radiolucent bone
- **DEXA scan** (BMD) = **gold standard if mod/high FRAX score**

***ALP** = marker of bone formation

**MANAGEMENT**[10]**:**

**Lifestyle:** **(if osteopenic** → repeat DEXA in 2–3y)
- Improve calcium intake
- Exercise (especially weight-bearing)
- Smoking cessation / reduced alcohol consumption
- **Medication review:** stop or reduce steroids if possible

**Physio:** reduce falls risk

**Medication:** **(if osteoporotic)**
- Vit D supplements – 400–800 U daily
- Calcium supplements – 500–1000mg daily
- Bisphosphonates – alendronic acid 70mg weekly

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[10] National Osteoporosis Guideline Group (2017) *Clinical guideline for the treatment and prevention of osteoporosis*

# Common adult fractures

## 'Terrible triad' elbow fracture

1. Posteriorly dislocated ulna
2. Fractured radial head
3. Fractured coronoid process

± Lateral collateral ligament damage

**Management:** surgical repair of joint capsule

## Colles' fracture

→ *fracture of **distal radius** with **dorsal displacement***

**Cause:** FOOSH (extended wrists)

**Epidemiology:** especially elderly women (osteoporosis → low impact #)

**Associated problems:** ulnar styloid #, median nerve damage / carpal tunnel, EPL tendon injury

**Specific initial Mx:** check function of median nerve / signs of carpal tunnel

**Further management:**

1. **Undisplaced:** dorsal splint then cast for 4–6w
2. **Displaced:** closed reduction
    - Bier's block anaesthetic + gas & air (haematoma block or sedation are also an option)
    - Traction & reduction to realign fragments
    - Placed in cast for 5–6w (check X-rays every 2w)
3. **Unstable fracture:** surgical fixation (percutaneous wires or plates)
    - If fracture **re-displaces** within cast
    - If dorsal radius fracture is **comminuted** (multi-fragmentary)
    - If **Smith's or Barton's** fractures

## Smith's fracture

→ *fracture of **distal radius** with **volar displacement***

**Cause:** FOOSH (flexed wrists)

**Management:** initial Mx as for Colles' PLUS surgical fixation

## Scaphoid fracture

→ *proximal (15%), middle (80%) or distal (5%)*

**Cause:** FOOSH / direct impact to hand

**Epidemiology:** all ages

**Symptoms:** pain & swelling in anatomical snuffbox, weakness of pinch grip

**Associated problems:** radial #, avascular necrosis (especially of proximal #)

**Investigations:** scaphoid series (PA, lateral + oblique + ulnar deviation)

**Management:** refer to orthopaedics as high risk of AVN

1. **Undisplaced:** cast including thumb for 8–12w
2. **Displaced:** internal fixation with a screw → risk of non-union = 5–10%

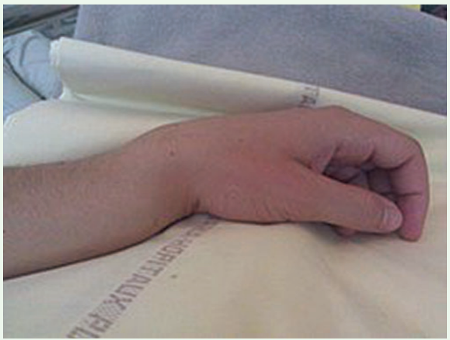
**Elbow dislocation** = 2nd most common after shoulder dislocation → due to FOOSH

**Radial head & olecranon fractures** = common due to FOOSH

**Avoid cast on elbow:**

**Instead use sling, or surgery with tension band wires or plates**
(joint stiffens too much in plaster)

**Fig. 19.17** Colles' fracture.



**Features of Colles' fracture:**

1. Dorsal translation & angulation
2. Flattened radial inclination
3. Radial impaction & shortening

Appearance known as 'dinner-fork' deformity

## Forearm fractures

*→ ulna and radius form a 'ring'* ***(normally breaks in 2 places)***

### GALEAZZI FRACTURE:

Fracture of distal radius + dislocation of distal radioulnar joint **(wrist)**

### MONTEGGIA FRACTURE:

Fracture of proximal ulna + dislocation of proximal radioulnar joint **(elbow)**

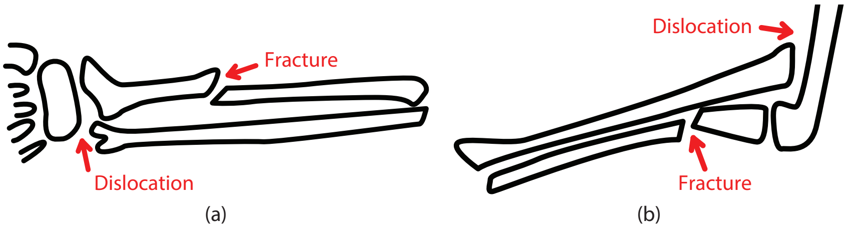


**Fig. 19.18** Forearm fractures: (a) Galeazzi; (b) Monteggia.

**Management:**
Open reduction & internal fixation

## Glenohumeral dislocation

### ANTERIOR DISLOCATION (96%)

**Cause:** fall on extended arm

**Epidemiology:** most common dislocation

**Signs:**

- Swelling & deformity (squaring of shoulder)
- Gap under acromion

**Complications:**

- Axillary nerve damage (shoulder flexion & abduction)
- Brachial plexus palsy
- Rotator cuff tears
- Recurrent dislocation (Bankart lesion / labrum damage)

**Investigations:** X-ray & MRI (assess rotator cuff tears)

**Management:**
Closed **reduction under sedation** (e.g. Hippocratic method)
+ check with X-ray & immobilisation for 1w in **cuff & collar sling**

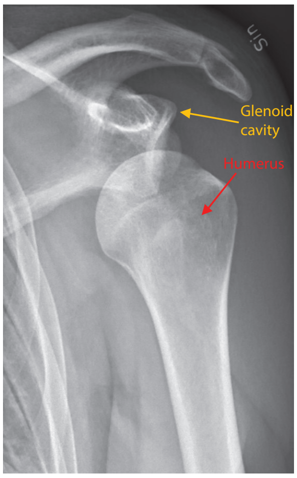


**Fig. 19.19** Anterior shoulder dislocation.

### POSTERIOR DISLOCATION (RARE):

**Cause:** may follow epileptic fit / seizure or polytrauma

**Signs: 'light bulb sign'** on X-ray

**Complications:**

- Locking → reduced ROM (esp. external rotation)
- Hill–Sachs lesion causing instability

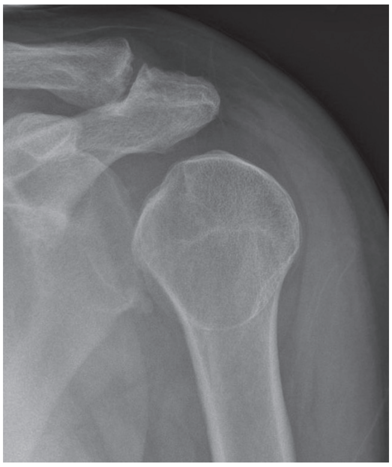


**Fig. 19.21** Light bulb sign in posterior shoulder dislocation.

**Bankart lesion:** damage to the labrum (usually anterior inferior part). If lesion affects the bone it is known as a 'bony Bankart' lesion.

**Hill–Sachs lesion:** dent in humeral head caused by impaction against the glenoid rim during dislocation.

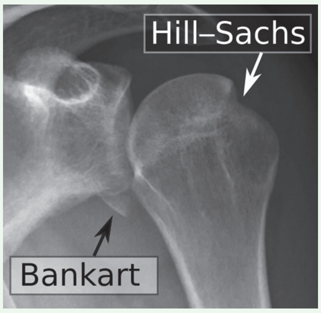


**Fig. 19.20** Hill–Sachs + Bankart lesions.

## Acromioclavicular dislocation

**Cause:** fall directly on to shoulder, contact sports, RTA

**Management:** depends on Rockwood grading
**Type 1 or 2:** sling for 1–2w then physio
**Type 3 or 4:** surgical fixation with a plate (type 3 can sometimes be managed non-operatively)

**Classifying AC dislocation: Rockwood grading**
**Type 1:** no joint disruption – AC ligament sprain
**Type 2:** <50% vertical displacement – CC ligament sprain
**Type 3:** 100% vertical displacement – AC & CC ligaments torn
**Type 4:** posterior displacement – AC & CC ligaments torn

## Clavicular fracture

→ ***common fracture in all ages*** *(5% all fractures)*
**Cause:** direct trauma or fall on shoulder

**Symptoms:** tenderness, visible deformity, tenting of skin

**Describing the fracture:** distal section displaced distally & inferiorly

**Complications:**
- Brachial plexus injury, vascular damage, skin necrosis
- Rib fracture & pneumothorax
- **Non-union** (1–5% risk → more likely if medial/lateral #)

**Management:**
1. Conservative: → collar & cuff sling for 4–6w + analgesia & rest
2. Surgery: → plate & screws or pin

**Indications for surgery in clavicle #:**
- Open fracture
- Neurovascular damage
- High risk non-union

## Humeral shaft fracture

*→ proximal (30%), middle (60%), distal (10%)*
**Cause:** direct or indirect trauma (high energy or if elderly with weak bones)

**Type:** usually spiral or oblique

**Complications:** radial nerve injury

**Management:**
1. Immobilised with cast 'U-slab'
2. Replace with brace after 2–4w

**Surgical management:** plate/screws or **rarely** external fixation

**Indications for surgery in humeral shaft #:**
- Open fracture
- Neurovascular damage
- Multiple trauma
- Displaced transverse fracture

## Proximal humerus fracture

→ ***most common fracture of humerus***
→ *described based on number of fragments*

**Cause:** falls → elderly women (weak bones) or high energy falls (cyclists etc.)

**Complications:** axillary nerve damage, brachial plexus palsy

**Management:**
1. Collar & cuff sling immobilisation + analgesia
2. **If younger / high demand:** screws/plate → risk avascular necrosis

**Risk factors for NOF #:**

- Osteoporosis
- Those at ↑ risk of falls

**Mortality of NOF #:**
10% at 1m
33% at 1y

**Complications of NOF #:**

- Avascular necrosis (AVN) of femoral head
- Non-union (5–30%)
- Dislocation (10%)

**Risk factors for AVN:**

- Irradiation
- Leukaemia & lymphoma
- Sickle cell disease
- Alcoholism
- Steroids
- HIV, CMV, hepatitis, rubella

**Only consider total hip replacement if patients:**

1. Were able to walk independently outdoors with no more than one stick
2. Are not cognitively impaired
3. Are medically fit for anaesthesia & the procedure

(hemiarthroplasty = alternative)

## Neck of femur (NOF) fracture

→ ***very common in elderly population*** *(especially females & those in institutional care)*

**Cause:** in the elderly following a fall

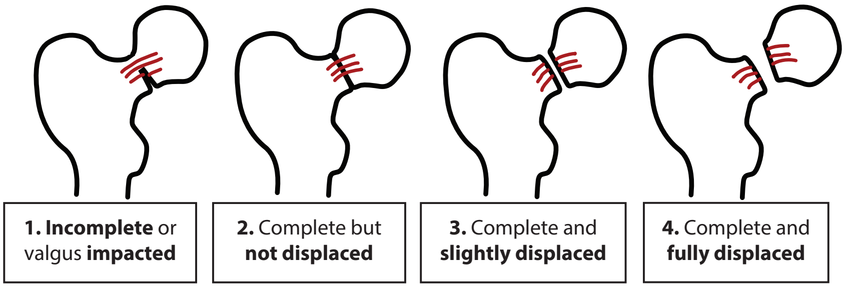
→ Important to assess for **comorbidities / complications of fall** e.g. AKI

→ Important to assess **baseline mobility** (determines Tx options)

**Classification:** intra- or extra-capsular

1. **Intracapsular fractures:** the Garden classification → based on **plain AP radiograph**

**Fig. 19.22** The Garden classification.


1. Incomplete or valgus impacted
2. Complete but not displaced
3. Complete and slightly displaced
4. Complete and fully displaced

Identify on radiograph by looking at **alignment of trabecular lines.**
(1 & 2 = difficult to spot)

**Specific management[11]:**

- Garden 1 & 2: blood supply intact → **cannulated screws** or 2-hole dynamic hip screws
- Garden 3 & 4: blood supply compromised → total or **hemiarthroplasty** (if fit & young **fix not replace** (with screws)

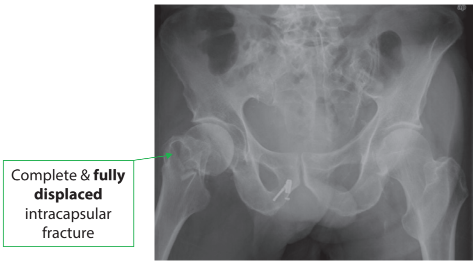


**Fig. 19.23** Type 4 Garden classification.

"1 & 2, fix with screws"
"3 & 4, will need more"

**General management:**

- VTE prophylaxis
- Analgesia – FNB or FICB
- Physio/rehab – weight-bearing recommended

2. **Extracapsular fractures:** → intertrochanteric or subtrochanteric

**Management:**

- **Intertrochanteric:** blood supply intact
  → 4-hole dynamic screws (or IM nail)
- **Subtrochanteric:** blood supply intact
  → intramedullary nail (DHS = too weak)

**Cause of subtrochanteric fracture:**

→ High impact trauma
→ 2° to lytic lesions (need CT)

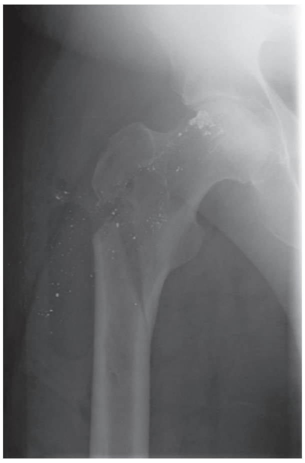


**Fig. 19.24** Subtrochanteric fracture.

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[11] AAOS (2021) *Management of hip fractures in older adults*

## Patellar fracture

**Cause:** 'dashboard' injury*, fall directly onto knee, direct blow, sudden quad contraction

**Associated problems:** tendon/ligament damage, avascular necrosis

**Management:** knee brace or surgical fixation (with tension band wires)

*occurs when knees hit dashboard in RTAs

## Tibial plateau fracture

→ *most commonly lateral plateau*

**Cause:** high energy trauma or elderly with weak bones

**Symptoms/signs:** pain, swelling & valgus/varus deformity

**Investigations:** X-ray + MRI → fat-fluid level on lateral X-ray

**Associated problems:** compartment syndrome & big soft tissue injury

**Management:**

1. **Undisplaced:** cast or knee brace for 6w
2. **Displaced:** internal fixation with screws/plates

## Tibial shaft fracture

→ ***usually spiral fracture*** *(caused by twisting force)*

**Epidemiology:** most common long bone fracture in adults (especially young ♂)

**Associated problems:** vascular injury, compartment syndrome & big soft tissue injury

**Management:**

1. **Undisplaced:** closed reduction + cast for 16w (or functional bracing)
   - Initially whole leg cast, then below knee cast after 4w
2. **Displaced/open:** interlocking intermedullary nailing
   - May need to start with external fixator device while swelling reduces

**Arteries at risk in tibial shaft #:** peroneal, anterior & posterior tibial (check pulses, CRT, temperature)

## Calcaneal fracture

→ *most common tarsal fractured*

**Cause:** fall from height, RTA

**Associated problems:** soft tissue injury, skin necrosis / blisters

**Signs:** shortened, widened heel with **varus deformity** + reduced Bohler's angle

**Investigations:** **calcaneal series:** lateral and axial (from below) views

**Management:** cast or internal fixation

## Ankle fracture

→ *classified with* ***Weber classification*** *according to relation to joint*

**Cause:** twisting motion

→ Skiers, footballers / high heels

→ Osteoporotic women

**Associated problems:** ligament disruption

**Signs:** tenderness, **extensive swelling & bruising**, inability to weight-bear

**Investigations:** X-ray – AP, lateral & mortise views

**Complications:** ankle osteoarthritis

**Management**[12]**:** may need external fixation first if swelling too extensive for cast/incision

↳ **Weber A:** usually stable

→ Cast/splinting for 5–6w

↳ **Weber B & C:** variable stability (often malleolar fracture & ligament disruption)

→ Closed reduction & refer for ortho review for possible internal lag screw fixation (with plates)

**Weber classification** (lateral malleolus / fibular fractures):

**A:** below ankle joint, with intact syndesmosis **(stable)**

**B:** at level of ankle joint (**stable or unstable:** check talar shift)

**C:** above ankle joint + medial malleolus fracture **(unstable)**

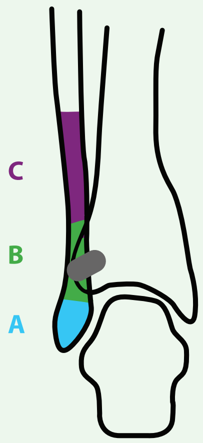

**Fig. 19.25** Weber classification.

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[12] BOAST (2016) *The management of ankle fractures.*

**Unstable ankle injury** =
deltoid ligament injury ± medial malleolar fracture
→ **X-ray shows widened syndesmosis + talar shift**

## Maisonneuve fracture

→ *fracture of* ***proximal fibula*** *+* ***unstable ankle injury*** *(talar shift)*

If notice talar shift on ankle X-ray, but no fracture, need to check higher up leg for proximal fibula fracture

## Pilon/plafond fracture

→ ***distal tibial fracture*** *due to talus being forced upwards into tibia*

**Cause:** fall from a height

**Complications:**

- Fractures to knee
- Vertical shear fracture of pelvis
- Vertebral fractures
- **Vascular damage:** anterior & posterior tibial, peroneal
- **Nerve damage:** superficial peroneal nerve

**Management: ATLS approach**

→ Temporary external fixation then internal fixation → poor prognosis (high risk OA)

## Wedge fracture of the spine

→ *a* ***fragility fracture*** *seen in the elderly*

**Cause:** fall from standing height in elderly (osteoporotic bones)

**Presentation:**

- Acute onset lower back pain (worse with movement)
- Localised tenderness

→ Multiple wedge fractures cause **hyperkyphotic deformity**

**Investigation:**

- X-ray
- CT – if unsure of stability
- MRI – if neurological signs

**Management:**

- Analgesia & early mobilisation
- **Kyphoplasty:** inject cement to stabilise
- Long-term osteoporosis management

## Traumatic fracture of C-spine

**Cause:**

→ **Young:** RTAs or extreme sports
→ **Elderly:** fall from standing

**Investigation:**

- **Full neurological assessment**
- X-ray (AP, open mouth, **lateral** ± swimmer's)
- CT – first line in trauma patients
- MRI – if neurological signs

**Management: ATLS approach**

- **Triple immobilisation** – hard collar, board & blocks
- Strong analgesia
- **Spinal surgery referral** to realign/stabilise fracture

# Common paediatric fractures

**Most common fracture sites in kids:**

- Distal radius (FOOSH) & hand
- Clavicle
- Tibial shaft, ankle & foot
- Radial shaft & elbow

## Incomplete fractures

→ Common in children due to softer / more flexible bones
→ Bones bend/compress rather than break

**GREENSTICK FRACTURES** = partial break in one side causes other side to bend
→ Common mid-shaft of forearm and lower leg

**BUCKLE/TORUS FRACTURES** = compression on one side of the bone causing bulging cortex
→ Commonly involve distal metaphysis of radius
→ Following FOOSH
→ **Appears as 'base of pillar' / bulge & often no fracture line seen**

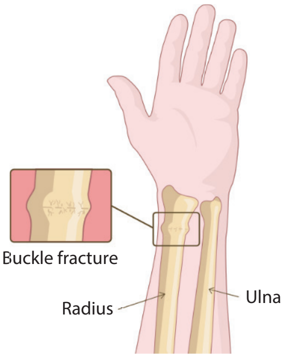

**Fig. 19.26**

**HAIRLINE (STRESS) FRACTURES** = small 'cracks' that do not traverse entire bone
→ Usually from overuse / repetitive stress-bearing motions
e.g. track runners, gymnasts, dancers

## Complete fractures

**COMMINUTED/MULTI-FRAGMENTARY FRACTURE** = bone broken into >2 pieces / crushed into fragments

**BUCKET-HANDLE FRACTURE** = fragmentation of corner of metaphysis
→ **Indicates non-accidental injury**

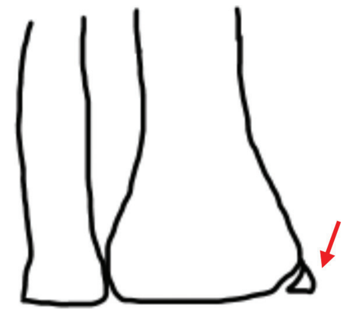

**Fig. 19.27** Red arrow shows 'bucket-handle' fracture suggestive of NAI.

## Growth plate fractures

→ Unique to children (common in growth spurt when physes are weakest)
→ Typically caused by great force during sports or playground accidents
→ Classified with **Salter–Harris system**[13] → determines Tx options

| Salter–Harris type | Treatment |
|---|---|
| I (S) | Splinting or casting |
| II* (A) | |
| III (L) | Open reduction & internal fixation |
| IV (T) | |
| V* (ER) | Surgery |

* Most common   * Likely to affect bone growth

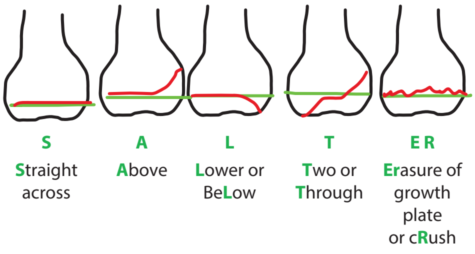

**Fig. 19.28** Salter–Harris classification.

## Management options for fractures

1. **Simple cast/splint**
2. **Brace/boot**
3. **Surgical fixation with metal pins/screws + analgesia**

---

[13] Salter R, Harris WR (1963) Injuries involving the epiphyseal plate. *J Bone Joint Surg Am*, 45:587

# Figure acknowledgements

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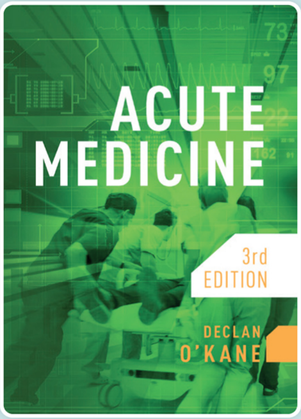
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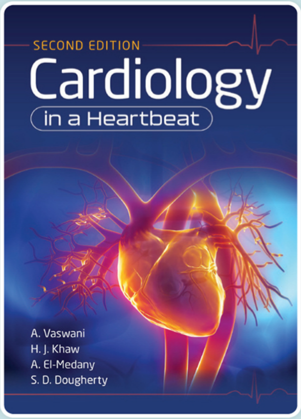
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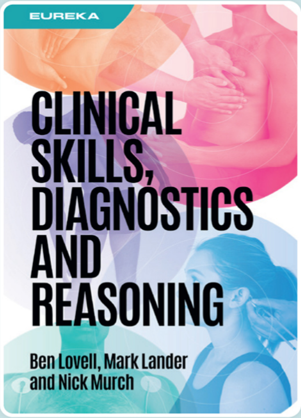
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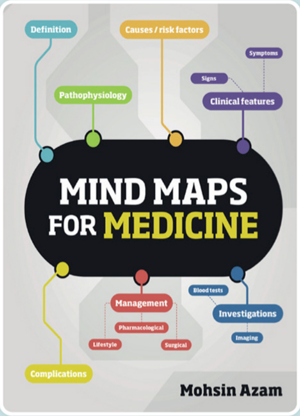
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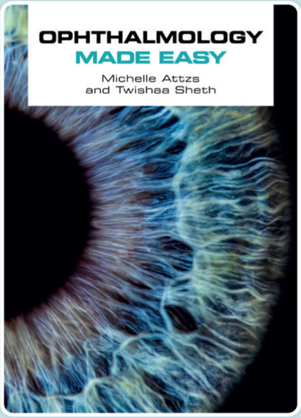
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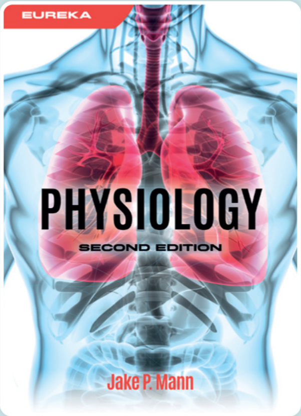
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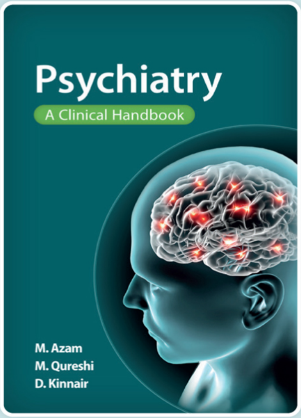
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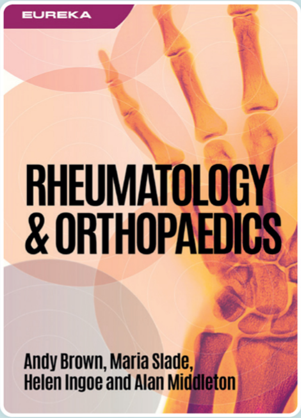
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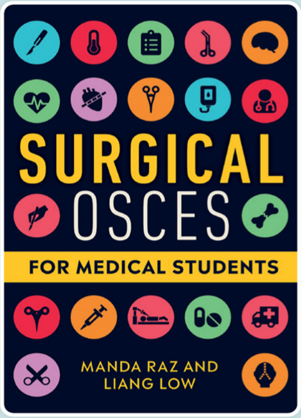
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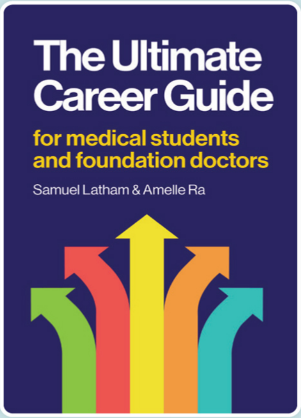
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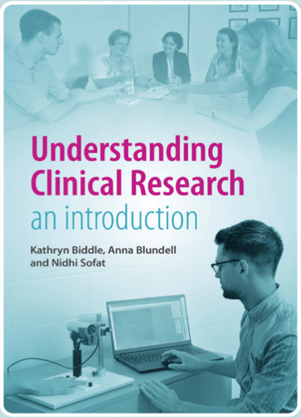
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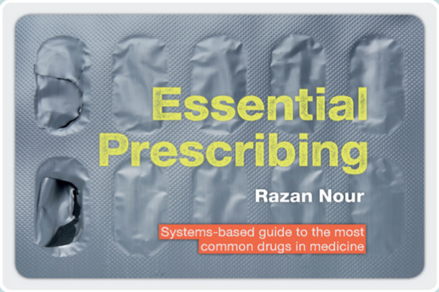
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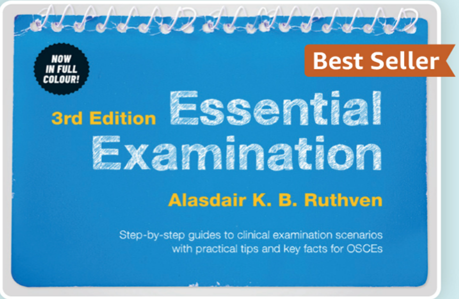
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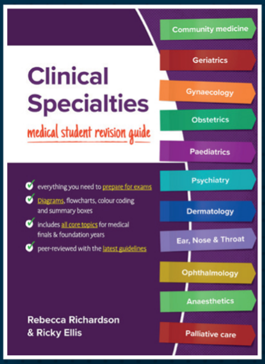

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## Student reviews:

*"These books have completely transformed my revision. The notes are so well organised and easy to learn from – very concise and colourful!"*

*"Used the **General Medicine and Surgery** for finals revision, my only regret was not having it earlier. The information is broken down in easily digestible chunks with colours and diagrams."*

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*"Amazing book which helped me pass into 5th year! A must for medical students."*

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